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LECTURES ON PATHOLOGY

LECTURES ON PATHOLOGY

DELIVERED AT

THE LONDON HOSPITAL

BY THE LATE

HENRY GAWEN SUTTON, M.B., F.R.C.P.

PHYSICIAN AND LECTURER ON PATHOLOGY AT THE
LONDON HOSPITAL, ETC. ETC.

EDITED BY

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AND REVISED BY

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EDITOR'S PREFACE.

In the end of last year Dr. Sutton decided to publish a revised and much enlarged edition of his "Medical Pathology," and entrusted the preparation of the work to me, intending himself carefully to revise all the lectures before sending them to press. Unfortunately, although a considerable number of lectures were in his hands, he had been unable to find time to revise more than four before his death; these are the lectures on Cholera and Diarrhœa, Typhus Fever, Small-pox, and Dysentery.

When, after Dr. Sutton's death, it was decided that the preparation of the book should be continued, Dr. Wilks kindly undertook the necessary work of revision. He wishes me to explain that his object has mainly been to ensure that the pathological terms are accurately used, inasmuch as errors might have crept in through repeated transcription. He has left the lectures, in matter and in manner, almost entirely in the form in which I sent them to him, this form being, as nearly as possible, that in which they were originally delivered.

The book has been compiled from several courses of lectures. Almost the whole of the original "Medical Pathology," published in 1886, has been incorporated in the present volume; it contained the course of lectures delivered in the summer session of 1885. In the summer session of 1886 the lectures were again taken down in shorthand by Mr. J. Cavendish Molson; these comprise Lectures VI. and VII., Lectures IX. to XX., and a portion of the lecture on Nervous Disease. The reports of these lectures are therefore full and accurate. The rest of the book is almost entirely derived from notes taken by myself in longhand, and written out usually on the same day; though not, of course, as complete as would have been a shorthand report, they are fairly full, and are transcribed as far as possible in Dr. Sutton's own words. The lectures of which I took notes were delivered in the summer and autumn of 1888, and in May 1891. Some of these lectures covered new ground, others dealt with the same subjects as those previously published, and have been used to revise and amplify these; for instance, about one-half of the lecture on Heart Disease, as now published, is taken from the "Medical Pathology," the rest from my own notes of lectures given in 1888; the two have been combined, so that they may be read as one continuous lecture.

The task has been a difficult one, and I cannot hope that the result will be entirely satisfactory, but I have earnestly endeavoured to do justice to Dr. Sutton's memory. His methods of description and illustration were extremely forcible, but often peculiar; nor had his phraseology always a close resemblance to that in common use. It did not, however, appear to me, and my view was shared by Dr. Wilks, that my duties as Editor demanded from me the attempt to put forth Dr. Sutton's teachings in a more conventional form. Had I done so, I might, perhaps, have pleased a few, but I should have displeased the majority of those who have come into personal contact with Dr. Sutton, and who will, I am convinced, prefer to have his teaching as it left his lips, imbued with his own peculiar and powerful individuality.

Nor would the attempt to recast the teaching have been successful. Those who knew Dr. Sutton well will know that in his mind were many

> " Thoughts hardly to be packed
> Into a narrow act,
> Fancies that broke through language and escaped,''

and that when his thoughts were given expression, it was in words which, though often peculiar, were chosen with deliberation, and which could not be changed to those in more common use, without entire loss of much which they were intended to convey.

Without doubt there is much that is here published which would not have been left in its present form by Dr. Sutton had he had an opportunity for revision; the free correction he made in the MS. of the four lectures mentioned above gives ample assurance of this; but it was impossible to know in most cases what he would have corrected and what he would have left untouched. Only in a few places, therefore, in which it appeared probable that Dr. Sutton's meaning, clear to Dr. Wilks and to myself, would be obscure to those who had known him less intimately, has some alteration in the wording been thought advisable. Otherwise, my duties have been almost entirely confined to transcription and arrangement; to removal of the redundancies and repetitions which necessarily occur in all lectures of this nature, more especially when delivered, as were Dr. Sutton's, entirely without notes; and to other trifling alterations inseparable from the preparation of spoken matter for the press.

Dr. Wilks' technical share in the work I have already explained, but I may take this opportunity of gratefully

In conclusion, let me say that the word "lecture," as used in this book, is to be understood as referring to subjects, not to matter given forth at one time. It was impracticable to conserve the true lecture form, nor would anything have been gained thereby. The division into subjects makes the book more useful for reference. The lectures on Heart Disease, Kidney Disease, and Nervous Disease are exceedingly long, but it did not appear to me advisable to break them up into such sections as "Dilatation of the Left Ventricle," "Strumous Diseases of the Kidney," "Hysteria," &c. A continuous train of thought runs through each of these lectures, although they deal with somewhat miscellaneous subjects. I have endeavoured, by the use of italics, and by the separation of paragraphs by spaces, to indicate change of subject, and render the lectures easier to read.

MAURICE EDEN PAUL.

ALDGATE, *October* 1891.

AUTHOR'S PREFACE

TO THE LECTURES PUBLISHED IN 1886.

TO THE STUDENTS OF THE LONDON HOSPITAL.

GENTLEMEN,

At the outset you would, I feel, wish me to express our united thanks to Mr. J. Cavendish Molson for his intelligent and patient industry in taking the shorthand notes of these lectures, and subsequently reproducing them; by so doing he made their publication possible.

In accordance with your desire, I have revised the notes, and whilst so doing have often felt that I should like to spend years in their revision; but I cannot complain, for I have been granted many years to gather what I have therein told you.

The manner of expression is inadequate. I doubt if any person will recognise that more fully than I do; but I may add that I have endeavoured to do my best.

These lectures were, as you know, delivered spontaneously, so that you might learn from their merits and their defects. That spontaneous character I have tried to retain, and only added and altered where I thought extension called for, to make them serviceable in your daily practice.

Permit me here to tender you my grateful thanks for the gentle and patient encouragement that you freely afforded me whilst I was delivering them; in so receiving them you helped me much—in fact, enabled me to deliver them.

Throughout, you will have perceived that I have endeavoured to show that we study Pathology—the science of disease—in order that we may find out the way to go and become medical men. As we travel on, we discern that we must take the course in its natural order: be men to become medical.

In studying Anatomy and Physiology we are allowed to learn that the human body is magnificently constructed for great usefulness—adapted for achievements to an almost unlimited extent; for its organised arrangements are designed to co-operate continuously, to progress continuously, and to be renewed with the elements and beautiful things of the outer world. So it is evident, and experience manifests, that success in Medical Art is attainable if the bodily conditions are allowed to go naturally.

In our labours we cannot fail to feel that the perfection of these means is beyond expression of admiration and praise; we can only hope that they will lead us to give their Constructor our reverence and love, in order that we may become gentle servants.

In seeing how wonderfully the living body is made to work exactly in accordance with the conditions of the outer world and universe, we are brought to recognise that we have ourselves, as medical men, to go the way to become correspondingly exact in our daily doings—to become able to teach how weakness and failure, by processes of orderly exchange, pass into strength and success. In fact, we are brought, and have to bring others, to see that success comes by failure, if the failure be properly regarded—the conditions really seen; for they are working together with the renewing elements around, thus interwoven with curative agencies.

Medicine, the curative proceeding, is an exact, because a natural, science. The dog licks his lacerated limb, and it heals; we, as human creatures, may even apply cold water continuously to our torn flesh, and it heals, as if in spite of our efforts; but to make our "medicine" an exact science, we must take to heart the truth that "he who would command Nature must obey her."

In our travail to become medical men, and at the beginning, we take a surface view: disease is considered merely symptomatically and nominally, what may be spoken of as dogmatic medicine. We must first perceive the surface signs—view the disordered superficies of things. And with plausible manner we endeavour to lead; but, as we travel further and deeper, and often take a header into the Slough of Despond, we endeavour to get free enough from surface entanglements to see into the circulations; then the plausible leaves us: and, as we cast our lingering regrets behind, we cannot but feel distressed that Plausible is handled so hardly by his friends for not knowing better than to go with us so far.

To reach the domain of perception and become reasonable, we must go through the rough—take the rough with the smooth—so that our eyes may be opened; otherwise, we are soon caught by Giant Despair, and we wander all our days amongst dead men's leavings; and we are not present when we are wanted and called for. To get our eyes open, to be men, we must not fear to make mistakes; the old teacher has urged upon us all not to be afraid to be fools, but to be fools for man's sake. Let us take to mind what the mountebank said in the beggar's kitchen: "The chiel that's a fool for himself, gude Lord, he's far dafter than I!"

Let us be fools, rough-and-ready as the treatment is, trenchant fools when needful. We have often to ask, "What is the use of being too serious when we are in the sea of difficulty?"—to float we must be easy.

In so doing we come to feel, and thereby to know, what is indispensable to get through disease; we gain the tension to enable us to proceed; but to possess that tension we must trust our senses to the utmost: it comes by contact. And I may add that I daily increasingly learn the value of what Lucretius first taught me: "In vain has man tried to prove that sense can ever err."

The senses can no more err than the reality can err; but in the working of the senses we, by our preconceived notions, are illuded and deluded by the shifting conditions and appearances;

and even old crafty counsellors are thus delayed by the sirens, for they do sing sweet lullabies. But if we go on trusting the senses, we come to perceive until we recognise that we vegetate and actually are animals with reason; that we are guided sagaciously and instinctively like animals, whilst we feel our way to reason through bewildering collections of disease and health.

So we come to know that by following the working of discord we are guided to harmony. As the engineer, by following the workings of friction, is enabled to divert and appropriate energy, so, by following the workings of disease, we are guided to know how to go for health.

We are thus led to show that a medical man has two great duties—to afford relief, and to guide persons to see how wonderfully capable in its functions human nature is, if it be naturally taken; and so endeavour to prevent disease and promote happiness. The very fitness of the bodily conditions reveals that happiness is the birthright of human creatures, and ancient teaching has shown beyond question that the soul is joyful in doing. All men long to be useful, and it is our business to guide them to succeed.

But success depends on unity. We, therefore, must work and feel as one in our own natures; be thoroughly one—thorough in our right, thorough in our wrong—until we come by progress to see the relativity of things, of truth. So rational medicine dawns upon us. We perceive that "there is nothing either good or bad, but thinking makes it so."

We are brought to recognise that disease in human creatures, as in animals and plants, comes from restricted views, untimely forcing, and limiting too much. If you would study that in Nature at large, look carefully into Darwin's great works. Amongst other treatises, read his teaching on "Animals and Plants under Domestication." There is collected a mass of evidence revealing that disease comes from too limiting interference.

So we discern that disease is the result of unrest, rest being helpful, because it is the timely taking of a position of safety. In disease, living creatures are not allowed to take up a restful position—that is, a position of certainty, a sure footing—and travel onwards according to their wants—natural happy progression; their feeling of ease is interfered with: and I have laboured to guide you to inquire how ease comes and is lost.

You will, I have little doubt, often be impressed and instructed, as I have been, by hearing and understanding persons exclaiming how delightful is the internal feeling of rest. Let the light guide you.

The Social Economist, perceiving how distorted are Society's proceedings, how baneful to order, has openly said, "Society prepares the crime and thus makes the criminal." Similarly with disease; and we need not look far in order to trace its antecedents and producers.

As we travel on and see how it is so, we come to recognise that disease, the distracted feeling in our wearied patients, is the outcome of society, and its individual disorder. However desperate the patient may seem to be, he may frankly tell us that we are mad and not himself, for he is doing his utmost to get out of the disorder—yet we are taught to recognise that he speaks truly. And as he struggles on fearfully through many tortures, and his agonizing sweats tell us how his tensions are strained, and we feel our own correspondingly strained, we come to realise how awfully cruel is the mistrust of human nature, what miserable failures are the systems invented to improve it. And I have had to say to the gentle mind, sitting sternly upright, silent in its madness, grim in its forbearance, "The grand thing is to go off our heads when it is necessary." We thus gain freedom, and come to see how worthless are the opinions we worshipped; how different things are from what we thought them; how much more sweet and merciful we ourselves become as we really know. And we are enabled to go the way and deny ourselves, because we have knowledge of poor self; and as we know ourselves, we trust others because we trust ourselves. And whatever tempestuous proceedings we may be coursing through, we may prosper and every day learn from others, and feel in ourselves, that—

> "As the morning steals upon the night,
> Melting the darkness, so their rising senses
> Begin to chase the ignorant fumes that mantle
> Their clearer reason. . . . .
> . . . . Understandings
> Begin to swell, and the approaching tide
> Will shortly fill the reasonable shores."

You will have noticed that I often quote Shakespeare, for he knew human nature.

I have learnt to see, as the "History of Civilization" teaches (Buckle), that the poet is the forerunner of the philosopher and the practitioner, because he is a maker by seeing how things are made and are enabled to progress.

H. G. S.

9 FINSBURY SQUARE,
September 1886.

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LECTURES ON PATHOLOGY.

LECTURE I.

INTRODUCTORY.

PATHOLOGY teaches the maxims of rational practice. If a medical man does not know pathology, he must practise empirically.

The practice of medicine was formerly based entirely upon empiricism, and to a large extent is still so based. Empiricism must necessarily form a large element in practical medicine; how far the medical man is guided to a deeper view than the purely empirical will depend on the amount of science, in the fullest sense of the word, which he possesses.

Medicine based purely on empiricism is a proved failure. You will ask me what I mean by success. In medicine a successful view is one that guides us to successful treatment. Now the empirics were not successful in the treatment of disease, and their disputes are proverbial. So many years ago thoughtful physicians came to say: "We cannot discard empiricism, since such knowledge as we possess is entirely based on it; but we need something more, we need knowledge, science, certainty. How are we to reach this? We must search and see into the processes of disease, we must not merely look at the surface, but must learn the deeper, inner processes of disease, and understand what we really have to deal with."

They considered a disease—pneumonia, for instance. Patients with pneumonia, it used to be said, could only get well if they were bled and salivated, but they found that many of the patients got well when treated with rest in bed and simple

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saline medicine. On the other hand, a great many died. Then they began to look beyond the lungs, and to examine carefully the bodies of those that died, and it came to be recognised that there are two kinds of pneumonia. In one class of cases, pneumonia occurs in people whose organs are comparatively healthy, and they usually get well without treatment. In other cases, there is pre-existent gross degeneration of tissue; it may be Bright's disease, or it may be the degeneration due to long-continued abuse of alcohol. Such patients generally die in spite of all our efforts.

In our examination of the processes of disease the first step is then to study morbid anatomy. But remember this, that you must find it to lose it. You must not go through life seeing all disease through the atmosphere of the dead-house, for morbid anatomy teaches only this, what disease has accomplished; what we see on the post-mortem table is past and gone.

We must then find it to lose it, but we can learn much from morbid anatomy. We learn that all morbid change—all involution—follows in the track of evolution. Wherever there is the most functional activity there is the liability to the greatest disorder. We learn that pathological changes are physiological changes in great excess. The physiological process is overdone and outwearied, and has become destructive from its very excess. It is a physiological state for the pleura to be moist; in pleurisy the pleura is excessively moist and we get a pleural effusion. The pleural secretion naturally contains a little albumen; in pleurisy that albumen is formed in great excess, and we have the formation of inflammatory lymph. It is needless to multiply examples, and is sufficient to say that all pathological changes have their physiological prototypes, and that all the physiological prototypes being useful, the pathological changes represent physiological function in abuse, in excess.

Morbid anatomy has further taught us this important fact, that diseased processes divide the body into parts, that by softening or by other change they make a separation. When people are passing into disease it is because they are losing the wholesome unity which constitutes health. In treatment we aim at restoration to unity. Morbid anatomy shows further that these local separations multiply and multiply in the body until death. The disease may begin in any part of the body, but as it increases, more and more local separations occur until death. However, whilst one part of the body is failing, other parts are doing their best to compensate for that failure—*e.g.*, if one kidney is destroyed the other kidney gets larger to compensate for that loss. The aortic valve becomes faulty, and the left ventricle gets much stronger. The pyloric end of the stomach is contracted, and the muscle of the stomach is much increased to get the food through the contracted orifice.

Having gained a knowledge of the grosser changes, we find that it is necessary to look deeper, and we take our microscopes to study the changes in the tissues. This study is called pathological histology. From it we learn that tissue degeneration always precedes organic destruction. In embryonic development the formation of a tissue always precedes the formation of an organ; and inversely, in pathological change, in morbid involution, we find that the destruction of tissue precedes the destruction of an organ. Here again we have evidence that involution follows on the old track of evolution.

We find that the microscope does not tell us enough, and we have to go deeper still, and we study the atomic changes in the tissues—pathological chemistry, this is called.

The stages we have then passed through are:

1. Empirical study of phenomena.
2. Morbid anatomy.
3. Pathological histology.
4. Pathological chemistry.

And what then?

I passed through all those stages myself, and they brought me to this, "I nothing know." But that was something, for this seeing our darkness is being on the road to light. Once a man finds out that he has missed the way, he is on the alert immediately to get into the right path. The examination of the body after death has been of immense service in removing ignorance and superstition, and has certainly brought us to be more charitable. None the less, this period, when he begins to realise his utter ignorance, is a most wearisome one in the student's mind. He is like a man lost in a wood; he cannot get out, but goes round in a circle, which continually brings him back to the point at which he first realised that he did not know the way out.

How are we to find out the way to the light? We must again carefully study the signs of disease at the bedside. It is true that the clinical signs of disease are merely surface phenomena, but these external signs reveal the functional activities within. I want you to get this fact firmly impressed on your mind, that the symptoms of disease are signs of the failure of functional activities. This, and what I told you above, that a pathological process is a physiological process in excess, are the two chief guides in the study of clinical phenomena.

To sneeze now and then is physiological, but if a man goes on sneezing persistently for half an hour, this is a morbid phenomenon, and we think that there is something seriously amiss; it may be that a cerebral hæmorrhage is coming on. It is physiological that on rising in the morning a man should hawk a little, to clear his throat from the mucus which has accumulated during the night; but if a man vomits two or three times whilst he is dressing, we know that this excessive irritability betokens that the physiological processes are disturbed, and we suspect Bright's disease, or brain disease, or some other serious organic mischief. Remember that extreme irritability always means weakness.

As the failure of functional activity increases, the symptoms get worse and worse, and their duration is ever longer, and at last the patient is never free from them. At first the symptoms which troubled him lasted a few minutes or hours, as the case may be; then there was a time when they lasted for days, but there were intervals in which he was quite free. But when the time comes that the patient is never free from the symptoms, we know that there is permanent organic disease. For instance, in cancer of the stomach situated about the lesser curvature, the tumour is behind the lower end of the sternum, so that it cannot be felt, and the symptoms are very obscure. The most prominent of all the symptoms is pain. The pain lasts ever longer and longer, and at last a time comes when the patient says it never leaves him, and we feel confident that there is organic disease of the stomach; soon we feel the tumour, which is so large that it is no longer covered by the ribs and sternum.

At the outset all disease is functional. This brings me to the definition of disease, and while I am defining I will explain also what I understand by the word pathology, since to teach pathology is the object of these lectures.

We define for the sake of thinking further and more clearly. What is disease? It is the condition of being without ease, it is the opposite of health. To say that a man is healthy means that he is going easy in everything. A healthy man does not know that he has a stomach. Of course he has been taught all about the stomach in school, but that is not knowledge. That part of his body is working easy, and he is healthy, in happy ignorance. Then he stays indoors too much, keeps out of the sunshine, he cannot assimilate his food, and gets a pain in his stomach—he is diseased. A German pathologist writes: "Scientifically speaking, we cannot say that any person is healthy;" and that is obviously true, but real education teaches how to go and to work most easily and happily. Pathology is "a knowledge of suffering and disease." The word is now often used as if it were synonymous with morbid anatomy, but I use it in the fuller and older sense that I have given you.

The great power of nature, unhindered by human ignorance, is always acting "easy"; this is seen beyond dispute as we study the human body in the construction of the joints, the serous surfaces, the heart and vessels, &c.; but we in our ignorance hinder the working and bring about uneasiness, disease, incapacity—and that is the origin of pain.

Health, then, is going "easy" in everything. It is going safely, without dangerous friction, because it is going with great ease. "We do that best which we do most easily."

Now, as I said above, at the beginning all disease is functional, and it is then that it is most obscure, but it is then that it is most important to recognise it. If we can recognise the onset of phthisis in this stage, before there is gross organic destruction of lung, we can cure it with ease and quickness. If we recognise in the early stage that Bright's disease is coming on, we can perhaps ward it off for years. There is a stage in which we can recognise rheumatic fever before the occurrence of arthritis; now rheumatic fever is essentially a disease due to excessive fatigue, and at this stage a week or two in bed may ward it off. And this applies to all disease, that at the outset it is purely a functional disturbance, and if we can recognise it early enough we can often ward it off. The advance of medicine will lie in recognising more and more the signs of functional failure, and we shall save people from organic destruction.

So you will understand more fully now what I meant when, in speaking of morbid anatomy, I said that you must find it to lose it, and that it is necessary to spend as much time in the wards as in the dead-house. Doctors who look too much at the dead side of disease are apt to give too gloomy prognoses, they have seen so much of what disease has done, and they cannot trust the human body. But every year I am more and more impressed with what the human body can do.

When we study the failure of organic function we are immediately led to ask, "What is the cause of this failure?" Now this is a most fascinating and rich study, but it is one in which it is easy to go astray. There are many causes of disease; we can never say there is one cause of disease; therefore it is more exact to say, "This disease has arisen in these conditions." As much as possible, in thinking about pathology, endeavour to get rid of the word "cause."

What are the "causes" of disease? See this clearly and never doubt. In the old days men used to say that disease was the work of an evil spirit—darkness with them always meant an evil spirit. A still gloomier view came in the Middle Ages, for it was thought that disease was a visitation from God. This was a view which left no hope, for a beneficent spirit which sent disease was no longer beneficent. These were the people who taught that life was a mistake and the body vile. And they not only held these miserable views themselves, but they handed them down to poison other people's lives with.

Then came John Hunter, and he taught that all disease and deformity is the result of interfering with natural processes. This is a view which gives us some chance of success amid all our failures. And in truth all disease is due to abuse—to extreme abuse, for to some extent abuse is allowed. If you tell a man he must never drink a glass of beer, you will probably make him a drunkard; if you want to make your child a liar, tell him always to speak the truth.

It is abuse of natural function—extreme abuse, and restraining, hindering, and interfering, that bring about disease. Every age of life is characterised by systematic interference. It is through our detestable systems that human misery comes about Interference is a mistrust of functional activity.

Once when I was with Sir William Gull, he said to me, "Sutton, human nature has never had a fair chance," and it is true. The more I see of the human body, the more I admire it for being able to endure so much and suffer so little.

The study of pathology reveals that one and all of the functional blessings of man are abused into disease.

"When we are born, we cry that we are come to this great stage of fools," says Lear. The mouth of the new-born child is stuffed with sugar and butter, its belly is cramped, and its legs are not allowed to kick. It has indeed come on to the "stage of fools." As soon as ever the new mucous membranes begin to absorb we meet with thrush and stomatitis, due to foolish ways of feeding. The child's functional activity increases. We keep it swathed in clothes, and do not allow it to go out unless we think it is a fine day; and when we send it out, we put it into a horrid machine. Then we are told that it is always getting cold and diarrhœa, and that it sweats and throws off the clothes at night—it has rickets. And so it goes on throughout life. It was the abuse of domestic life which wrecked the civilisations of Asia, and which may wreck ours too.

Failure of function leads to organic destruction. It used to be thought that structure makes function, but it is the reverse—just as the blacksmith makes the horseshoe, fitting the iron for service. In nature it is evident that all function is making union—"a living and a loving"—while in disease there is disunion.

Disturbance of function precedes any structural decay, therefore study symptoms in the wards and observe fixed condition in the post-mortem room. In symptoms we may observe a swelling, a vomit, or other definite outpouring; but there is also to be considered a ceaseless rolling on—a circulation within a circulation—the salines within the colloids, the air within the water, the heat and light energies within all, finer and finer still. In symptoms we have the signs of diseased disturbances in these media. The circulation is ever changing and infinite. There are therefore two great effects always to be noticed—the living and changing, and the dead and more fixed signs.

Inflammation is a disturbance of this ceaseless circulation, passing into, through, and from the more fixed tissue-forms, and from the study of this disturbance we learn much of what diseased processes really mean. Therefore inflammation must occupy much attention.

The aim of a medical man is to restore ease, hence he learns pathology, and he does not overlook the subjective any more than the objective symptoms. If he stay in empiricism, he sooner or later finds to his sorrow that, whilst opportunities are fleeting, experience may be fallacious.

LECTURE II.

INFLAMMATION.

ALL pathological changes are merely modified physiological changes, there is no essential difference between the two. Thus, serous effusion from the capillaries into the plasma of the tissues is an ordinary process of healthy nutrition, but that serum in excess becomes dropsy. White corpuscles coming out of the capillaries into the plasma is also a normal process, but white corpuscles in excess become the products of inflammation and suppuration. In normal tissue-change the colouring matter of the bile and of the urine are derived from the red blood-corpuscles. In disease we may see processes like these take place. Red blood-corpuscles appear in the urine, a little while afterwards they are broken up and are replaced by hæmatin granules, and later these granules are replaced by the normal pigment of the urine. A person may at first complain merely of nervousness, and successively of tremor, of spasm, and of epileptic fits. These gradations show how insidiously healthy pass into morbid processes.

The tissues consist of tissue-forms and circulating material. A common prelude to inflammation is a morbid fixity of the tissue-forms. That which we know as death is the fixed forms remaining, but the circulation has ceased. So long as the circulation continues there is life ; that applies to the part and to the body as a whole; and just as the circulation in the tissues is maintained by the general circulation of the body, so the latter is maintained by the circulation of the deeper and outer world. As in the struggle for existence there is a continual exercise and mental influence, so there are similar changes in the nutrition of the tissues, in combining with the water that holds in suspension all that they require. This union is kept up by what is known as "vis nervosa." The connecting actions between the circulating material and the fixed tissue-forms is always rhythmical, *i.e.*, regularly repeated, and the blood circulation is carried on by light and heat, and both are rhythmical.

Inflammation has long been recognised by redness, heat, pain, and swelling; and when these qualities were found the part was said to be inflamed. What is the redness due to? To the vessels being excessively charged with red corpuscles. What is the pain due to? To the surrounding tissues being over-distended, and the nerves pressed upon; the increased tension gives rise to the pain, and when it is relieved the pain usually subsides. What is the swelling due to? To the accumulation of blood in the vessels and to the pouring out of fluid from the blood. What is the heat due to? The heat is due to the excessive accumulation of energy in the part, an excessive accumulation of power which, if it does not get vent, will destroy. Inflammation essentially consists in this, that there is a dangerous accumulation of energy in a circumscribed part which must get vent, or destroy. In inflammation the first appreciable change is hyperæmia—the blood-vessels become excessively charged with blood. The next thing that happens is that colourless blood-corpuscles collect about the endothelium while the red blood-corpuscles roll on in the centre of the vessel. The next thing that is noticed is that the colourless corpuscles have passed through the walls of the vessels and are seen outside them amongst the tissues, so that the tissues become cloudy from the excessive accumulation of blood-serum and colourless corpuscles. If the inflammation goes on still further, the red corpuscles also gradually ooze through the vessel wall, the circulation of the red corpuscles in the centre of the vessel gradually ceases, and there is complete stagnation in the vessels, and they die, being plugged by dead blood-clot.

To account for these changes some have said that there may be a spasm excited in the wall of a small artery, due to disturbance of nervous power, and that this spasm is followed by paralysis of the wall of the vessel and excessive distension.

Others deny the preliminary spasm, and say that the vessel is paralysed from the outset. There is a basis of truth in these views, but they are imperfect in that they do not consider how in healthy nutrition the contents of the vessels and the plasma of the tissues continually and evenly work together, whereas in inflammation there is failure of this co-operation, and this leads to the outpouring of serum and corpuscles into the tissues.

In inflammation the dilatation of the blood-vessels may be overcome and the vessels may pass into their normal condition, the exuded colourless corpuscles break up into a granular material, the serum passes on into the lymphatics, and the red corpuscles break down into a brownish-red pigment. As this takes place the plasma of the tissues recovers its normal conditions; there is recovery from inflammation.

In other cases, however, the vessels do not recover their normal size, they go on pouring out serum and corpuscles, and the tissues around swell and soften. Inflammation softens all tissues, bone, muscle, cartilage, and nerve, so that whenever you think of inflammation, think also of softening. How far can this softening go without complete destruction? The tissues will certainly be destroyed if there is continually increasing swelling, and therefore our first duty in inflammation is to relieve tension. If the process of softening be slow it is usually termed ulceration—a slow crumbling down of the tissues; and as the crumbling down goes on we can see a sweat of serum through the part, and with the serum come also colourless blood-corpuscles. This is called pus. I have told you that this process is only an excess of the physiological process of exudation of serum and corpuscles from the vessels. Is there a healing process in inflammation as well as a destroying process? Certainly there is, and the old surgeons used to say, "Wounds heal by inflammation." Why then do not all ulcers heal? Because the colourless blood-corpuscles perish before they can develop into living tissues; they become granular and fatty, and die. Why does not the serum live? Because it flows out and cannot be saved. The healing of an ulcer consists in our management of these two factors, the corpuscles and the serum. Salves, Liston's cold water, and other dressings, act as the conservators of these two agents. The slow crumbling away of the tissues is then called ulceration or caries, the tissue is eaten away; but if, as the circulation ceases in the vesels, the surrounding tissues are excessively stretched by the accumulation of inflammatory products, large areas of tissue may die—that is, there is sloughing or gangrene. If such tissue is packed with white corpuscles, there is commonly suppuration combined with the sloughing. It is very common also for the red corpuscles to ooze out in smaller or larger quantity, and as we know that a higher tension in the vessel is required to cause the red corpuscles to flow out, we know that when this occurs there is a greater inflammation. Therefore the most severe and rapidly fatal forms of inflammation are accompanied by much hæmorrhage, and hæmorrhage and sloughing commonly occur together. As illustrations I may mention hæmorrhagic pleurisy, pericarditis, and peritonitis, which are very severe forms of inflammation. In scurvy, in which there is much tendency to hæmorrhage, inflammation is a most dangerous condition.

If we understand inflammation, we know a great deal of pathology. I repeat that inflammation essentially consists in the vessels becoming extremely engorged, and the blood becoming more and more stagnant in the vessels, there is failure of the normal interaction between the blood in the vessels and the plasma of the tissues, and there is an outpouring of serum and corpuscles from the vessels. What are the conditions which bring about congestion—that is, dilatation of the vessels? To obtain clear views, think of what we have to deal with in a vessel, and how the circulation is carried on.

A vessel is a tube made up of plasma, muscle, and nerve. The contents of the tube are blood. The plasma of the blood is connected through the plasmic substance of the vessel wall with the plasma in the tissues, and this, on the other hand, is similarly connected with the contents of the lymphatics; and in thinking of the circulation we must not confine our ideas to the blood flowing on through the vessels, but must also keep in mind the flow from the blood into the tissues and back again, and from the tissues into the lymphatics, and we must think of the onward flow of the lymph-stream. The blood flows on in the vessels because there is a regulated pressure sending it into them, and an aspiratory force drawing it out again, and in health these forces maintain an even and, within limits, uniform tension within the vessels. The force that sends the blood along the vessels is derived from the heart, and the exercise of this force, and the activity of the muscular vessel walls, are controlled by the vaso-motor nervous mechanism. The force drawing the blood out of the vessels is derived from the respiratory mechanism and from the right side of the heart. This keeps the veins and lymphatics clear, so that the blood and lymph flow freely along them ; it is assisted by the pressure on these vessels which is exercised from outside during the contraction of the muscles amongst which they pass.

What are the "causes" of inflammation? Venous congestion is one of the commonest, long-continued and extreme overfulness of the veins ; and whenever there is venous congestion, it is always an anxious inquiry as to how far the distension of the veins and the attendant œdema will go before giving rise to acute inflammation. There is some failure in the lungs or right heart, or some pressure or disease in the veins, so that the outflow from the vessels is hindered and they swell. In this state trifling or imperceptible additional causes give rise to inflammation. Illustrations of this are : varicose veins, leading to venous congestion of the skin, followed by œdema, inflammation, ulceration, and suppuration, and maybe even sloughing; venous congestion of the pharynx, followed by œdema and inflammation ; venous congestion of the lung or kidney causes œdema, and this goes on to inflammation, and in this way arise some forms of pneumonia and nephritis. In this connection we think of the acute nephritis that is liable to come on in the course of fevers and in cases of lung and heart disease. Myelitis is also liable to come on when there is great venous congestion of the cord.

The opposite condition of deficient supply of blood from the arterial side is also a factor in inflammation, as when the blood pressure is persistently lowered from some cause, or when there is disease of the arterial walls. This is not so important as venous congestion, with which, however, it is often combined, as in fevers. In fevers there is another condition which predisposes to inflammation.

An unhealthy state of the blood is a common cause of inflammation by leading to imperfect nutrition of the tissues. We at once think of "blood-poisoning" in the various contagious diseases—syphilis, typhus, scarlatina. In these we are led to believe that there is some death in the blood itself, and some corresponding death in the tissues antecedent to the inflammation. There is another class of cases in which the poison originates within the body, as in the so-called uræmic conditions, and where the tissues are poisoned with bile. Either of these states may lead to inflammation. In some cases poison comes into direct contact with the tissues, as when peritonitis is set up by perforation of the intestine, of the gall-bladder, or of the urinary bladder. Some of the most fatal forms of inflammation arise in this way.

An excessively watery condition of the blood commonly leads to inflammation; and a loss of red corpuscles, which causes anæmia and hydræmia, has a similar effect, hence inflammation often comes on after hæmorrhage. Some unhealthy change in the salts of the blood may lead to inflammation, as instanced by inflammation in scorbutic and purpuric states. A morbid change in the albuminous constituents of the blood, as an excess of fibrin, has long been recognised as a cause of inflammation. An excess of colourless corpuscles in the blood may be attended by inflammation, as in scrofula, in leucocythæmia, and in lymphadenoma. It was formerly thought that an excess of red corpuscles in the blood, what was termed plethora, gave rise to inflammation,

Another extremely common condition producing inflammation is degeneration of the tissues themselves, or, as I put it at the beginning of the lecture, a morbid fixity of the tissue-forms, leading to obstruction to the circulating material, and hence imperfect nutrition of the tissues. This is how fibroid degeneration brings on inflammation. Fibroid degeneration of the kidneys leads to nephritis, fibroid degeneration of the cord to myelitis, fibroid degeneration of the lung to acute pneumonia. Many more instances might be mentioned.

Morbid growths are another cause of inflammation; an example is tubercle.

Some violent perturbation in the vis nervosa, such as an injury to the sciatic nerve or to the spinal cord, may give rise to inflammation. It is a similar kind of nervous disturbance which gives rise to the inflammation known as herpes, which follows the track of a nerve. When herpetic disturbance is coming on there is at first in many cases pain, which manifests a disturbance in nervous energy, and the inflammation follows. Another instance of inflammation due to disorder of nervous energy is arthritis in connection with disease of the spinal cord, and this may be difficult to distinguish from rheumatic inflammation; instead of a joint the skin of the leg may be affected in such a case, and it may be difficult to distinguish it from erysipelas, or the dermatitis may be vesicular or bullous. In urticaria, due to disordered nervous conditions, there is a congestive œdema of the skin, which in severe cases passes beyond this into inflammation. The insane are very liable to inflammation, and to very peculiar forms of inflammation, so much so that it has been said, "The flesh itself is mad." Their lungs, their stomachs, kidneys, joints, or organs of sense may undergo destructive changes. Mental disorders are then commonly associated with inflammation, and mental shock may bring on acute pneumonia. Pneumonia and herpes of the lip occur together after great shocks and strains of nervous energy. Other instances of the association between nervous disorders and inflammation are, the endocarditis seen in chorea, the pericarditis liable to come on in Addison's disease, and the various forms of inflammation that come on in epileptics.

A common cause of inflammation is mechanical injury, by which the tissues are bruised, lacerated, or concussed. By concussion we mean an exceedingly fine molecular change in the tissues, due to violent shaking. As an illustration of concussion: a man was caught in some machinery, whirled round, and brought dying to the hospital, and at the post-mortem examination there was neither external nor internal marks of injury; but on microscopical examination we found an enormous amount of molecular change and exudation in the tissues of the brain and other parts. That was simple concussion. Under mechanical injuries we may also class burns. Burns produce inflammation in two ways, Mechanically we have the direct action of the heat, swelling up and destroying the tissues, as we can see in the skin, and in the burned region severe inflammation ensues. But burns act in another way on the skin; by disturbing the nervous energy, and by arresting respiration, they lead to venous congestion and inflammation of the viscera. A lad was admitted to the hospital with a burn and was apparently doing well, when suddenly enteritis came on, and he died from the severe diarrhœa.

It is usual to say that extreme exposure to wet or cold, or great variation of temperature, may produce inflammation, but this, except perhaps in the case of mucous or catarrhal inflammation, of which I shall speak later, does not occur if the body be healthy. Healthy persons are exposed to all extremes without getting inflammation. So long as the tissues of the body are not dying, they can withstand extreme changes in the elements.

Experience has shown beyond all question that acute inflammation does not supervene from such causes in the healthy body. What are the facts which have led to this conclusion? It has been noted for many years that when death results from acute inflammation there has been antecedent disease, and the exceptions have been so few that we cannot but consider that in these we have overlooked the antecedent disease. But in all chronic failure of health acute inflammation is commonly met with.

Lastly, in considering the conditions which produce inflammation, remember that they never occur singly, but several of them act at the same time.

What forms does inflammation take? Remember that the body is made up of three distinct layers. There is an outer, which we will call mucous; there is an inner, which we will call serous; there is a middle, which we will call vascular or connective.

The outer or mucous inflammation differs in many features from the inner or serous inflammation. First of all, we have to recognise that the mucous surface is much more exposed to vicissitudes, to injurious external influences, than the serous surface, and therefore mucous inflammation is a much more common form of inflammation. The pleura, peritoneum, and pericardium are not so open to irritation, and therefore, whenever there is inflammation of these membranes, more severe causes have been in operation.

A pathological process is a modified physiological process. A mucous membrane is made up of a thick layer of epithelial cells and a comparatively thin layer of connective tissue underneath; it is a membrane mostly composed of cells which may be readily detached. If these cells are put under the microscope it is difficult, under some circumstances, to distinguish them from pus-cells; and, as a matter of fact, in inflammation of mucous membranes pus is very readily formed. The pleura, peritoneum, and pericardium are covered by an exceedingly thin layer of cells, but have a thick layer of fibrous tissue beneath the cells. In the inflammation of the serous membrane also a substance like to the inflamed tissue is thrown out; a fibrinous exudation is formed, which is readily modified into fibroid tissue, and may form adhesions or thickened membranes. These distinctions are not absolute. We sometimes meet with purulent inflammation of a serous membrane, and on the other hand with fibrinous exudations on the surface of a mucous membrane, but generally speaking there is the broad distinction I have given you between the types of inflammation of these two surfaces.

Mucous inflammation commonly takes the form of catarrhal inflammation, and usually results from changes in the seasons and extremes of heat and cold, and therefore often appears in spring and autumn. Examples are, laryngitis, catarrhal enteritis leading to diarrhœa, gastritis, catarrhal jaundice, tonsillitis, and various catarrhal inflammations of the skin, as eczema. In all these forms, with the exception of the last, we note that there is an excessive discharge of mucus, and this mucus becomes mixed more and more with colourless corpuscles from the blood. The more admixture of corpuscles there is the yellower becomes the secretion, it is purulent; but we notice also that the epithelial cells themselves swell up, and that their nuclei multiply; they swell so much that they burst, and the epithelium becomes more and more detached.

Such is inflammation of a mucous membrane. There is only a difference of degree between catarrh and catarrhal inflammation; the latter is a more severe process, and there is a greater exudation of leucocytes. Since the epithelium of a mucous membrane so readily swells up and becomes detached, ulceration of mucous membranes is common. On the other hand, if ulceration occur in inflammation of a serous membrane there is some very destructive condition at work. Inflammation of a mucous membrane may be attended by a streaky blood-stained discharge, but there is not usually much hæmorrhage. Where much hæmorrhage does occur, it is because some considerable vessel has been opened by an ulcerative process, or because the vessels are diseased. If the inflammation of a mucous membrane is very severe it extends into the sub-mucous areolar tissue. This is the great danger of inflammation of a mucous membrane, because if it extends deeply into the parenchyma, so that the blood-vessels become extensively plugged, the mucous membrane is liable to perish, and gangrene to come on. An example of such gangrene is seen in noma, which attacks the gums and cheeks of children.

A mucous membrane extremely rarely becomes adherent as a result of inflammation, whilst after inflammation of a serous membrane adhesion is exceedingly common. Further, in inflammation of a serous membrane we find fibrin usually, not pus; ulceration is rare, and gangrene exceedingly so.

There are three morbid conditions that we are continually meeting with in connection with inflammations: these are, anæmia, scrofula, and rickets. In the following lectures we shall study these diseases, which are very common in crowded towns.

LECTURE III.

ANÆMIA.

THE chief characteristic feature of anæmia is deficiency of the red corpuscles of the blood. Now these corpuscles give strength to mind and body, give strength to human nature, and therefore one of the most common symptoms of anæmia is weakness. Patients say, "I feel so weak, and I am so incapable of doing what I used to do with ease." With loss of strength there is loss of power for enjoyment. There is disturbance in many physiological functions: digestion becomes weak, with fulness, flatulence, discomfort after food, so that many of these patients come to the doctor complaining of dyspepsia; the function of the bowels becomes weak, so that constipation is very common. In some it may alternate with diarrhœa, which may be well-nigh uncontrollable, there being a tendency to catarrhal inflammation of the bowel, which may pass into dysentery, but simple constipation is more common. The function of the brain also becomes disturbed; not only does the patient feel languid, but he has difficulty in carrying on processes of thought; it is difficult for these patients to hear and to see, for the sense functions of the body are weakened. Young women may have perverse appetites. As brain function becomes more disturbed neuralgia is common—neuralgia of the trigeminus, gastralgia, neuralgia of the sacral plexus, backache, neuralgia over the lower part of the abdomen, pain over the region of the heart, intercostal neuralgia. There may be irritability of the bladder. There is increasing tendency to nervous and catarrhal trouble, mucous exudation from the throat and tongue, and catarrh of stomach with vomiting. Not a few about this stage pass into melancholy;

their brains become confused, they care for nothing, and have to be put into an asylum. If there be any tendency to epilepsy, the disturbance may take the form of fits; if to rheumatism, that of rheumatic fever; if to phthisis, phthisis may come on. Therefore, in every case of anæmia we have to think of what is the family disease-tendency, and treat the case accordingly. During this stage uterine function becomes much disordered, and amenorrhœa supervenes. A patient with anæmia had melancholia; she still menstruated, but the menses were pale; the melancholia passed away as their colour returned. Leucorrhœa may come on; very commonly the uterine ligaments and muscles lose their tone, and there is some amount of displacement. Respiration fails, shortness of breath comes on, and associated with this failure is loss of heat. Patients complain of cold hands and feet. There is increasing failure of brain, until silence becomes a marked feature, and there is great disinclination to move, because, the patient says, "It tries my breath so." Muscular weakness and breathlessness are the most marked symptoms of anæmia. The weakness may lead the patient to lie in bed, and it may increase until he dies. We find, post-mortem, that all the organs are pale—the brain, spinal cord, lungs, liver, and kidneys, all pale, and we may take a clot from the right side of the heart and find it exceedingly pale.

With diminution of red corpuscles there is danger of coagulation of the blood; therefore anæmia and thrombosis are often associated, thrombosis most commonly of the leg, but it may occur in other parts. Therefore, when anæmia is present, and there is aching in the calf of the leg, at once make the patient go to bed. Have this case in mind. A young girl was at school, and became very anæmic; then she began to cough, and other chest symptoms came on, and pneumonia. She died from thrombosis of the pulmonary vessels. A patient had been suffering from anæmia for some time, with pain in the head, and other symptoms supervened. She died, and we found thrombosis of the vessels of the pia mater. We see this tendency to coagulation of the blood within the vessels alike in chlorosis, and in the so-called pernicious anæmia. In pernicious anæmia there is fatty degeneration of the muscle of the left ventricle, and it is this which usually kills the patient; the heart's power grows weaker and weaker, and its action more and more irritable, so that one of the most distressing symptoms is palpitation. The fatty degeneration is usually detected by yellow zigzag markings across the columnæ carneæ of the left ventricle. We find such markings after death from all kinds of severe anæmia.

Œdema is connected with anæmia; therefore œdema of the feet comes on in bad cases; the increasing œdema renders the patient extremely liable to inflammation.

Experience has shown that no one cause can be assigned for anæmia; the "causes" are multiple. It may attack a girl a year or two old and kill, and afterwards attack a sister of that child and kill; there is something in family tendency. Again, it comes on at particular ages, whenever the body has to undergo some great change, or when the mind has to become specially active. It comes on when children go to school, or it appears at the onset of menstruation. Again, it comes on when the body is about to enter upon arduous work, and is often the forerunner of phthisis and other diseases of adolescence. "Young men to labour; middle-aged men to give counsel; old men to pray," the Greeks used to say. Anæmia comes on when certain stages of life are passing. Fatal idiopathic anæmia often comes on in the middle periods of life, or in the very aged. "He came to his end like a sheaf of corn—he was ripe." "He had a long life, a merry life." You will see aged patients getting paler every year. As old age comes on, fire and wine are wanted.

LECTURE IV.

STRUMA.

In struma it has long been recognised that there is general weakness of nutrition, and that many constitutions may be characterised from birth to old age by such weakness, by tendency to œdemas and local congestions leading to inflammation. Such is the strumous constitution. Strumous changes may occur in any organ of the body, in the brain, the spinal cord, the testicle, the lungs, the kidneys, the liver, the spleen; such changes are more common in children than in adults. They are especially common in lymphatic structures and glands. In this lecture I shall describe the general characteristics of struma, and the typical strumous inflammation of the lymphatic glands, and shall also consider strumous disease of bone and of the testicles. An account of strumous disease of other organs, the kidney, the larynx, the brain, &c., will be given in other lectures.

In the lecture on Inflammation I spoke of the circulating plasma and the tissue-forms. The foundation of animal nature is plasma. The fundamental affection in struma is disorder of the circulating plasma. This circulation is carried on by the energy of heat and light, and the old writers were right in connecting darkness and scrofula. A watery milky fluid containing corpuscles passes from the plasma into the lymph-spaces, and thence along the lymphatics into the lymphatic glands.

The plasma gives in and takes out; in other words, it is elastic—it receives and transmits a certain amount of power, and it is healthy so long as it keeps its elasticity. One of the great characteristics of the strumous condition is that there are local failures in the circulation of the plasma. As the plasma loses its elasticity there is an increasing tendency to local œdema —the serum is not sent on into the lymphatics—there is an increasing tendency to venous congestion—the tissue swells, and takes on acute inflammation, and that inflammation recurs until it disintegrates the structure. Strumous conditions are distinguished by their tendency to recur, and by the blueness of the skin. For example, strumous disease of the throat easily takes the form of enlarged tonsils, recurring congestion of the throat, and repeated tonsillitis; so that medical men have regarded enlarged tonsils as indicative of a tendency to phthisis. With these enlarged tonsils there is a corresponding weakness in the lymphatics of the neck, so that the glands commonly become enlarged and take on inflammation. The strumous inflammation is very insidious, and it has a peculiar manner of burrowing. It is often found necessary to insert a blunt-pointed bistoury under the mined skin and slit it up, so that the wound may heal up from the bottom.

I will now describe strumous disease of the glands. One of the early changes in strumous disease of the glands is swelling. In some patients it comes on so insidiously that there is neither pain nor tenderness, but it is noticed that other adjoining glands are becoming similarly affected; the swelling has a remarkable tendency to spread. The swelling is elastic, and we regard it as an œdema of the gland. The disease may subside in this stage, especially if secondary to throat affection. On the other hand, it may go farther, and the outline of the gland is lost. The extended swelling around reveals that the cellular tissue about the gland has become œdematous; later the part becomes red, and we know that acute inflammation has set in. It gets more and more red, and the redness is attended by fluctuation, so that we know that suppuration has occurred. The tendency is for the pus to be discharged externally, and over the part where it is going to break through, the red skin shines from pressure. Much of the gland may be destroyed by repeated suppuration in this way. The repetition of the suppuration is a most striking feature; although the gland may be mostly destroyed, there commonly remains some caseous matter which sets up further inflammation. Some of the deposit is apt to be converted into calcareous matter which may soften down, and be discharged through the skin. The removal of this calcareous matter is a curative process of the inflammation. There may have been for many years futile attempts to heal these sinuses before the dead accumulations were removed.

What is the appearance of a strumous gland when cut into? It may be extremely red and congested, but this is comparatively rare. More commonly it is pale, exceedingly anæmic-looking, tightly packed within the capsule, feeling firmer than is natural. On section we see among the pale material some congested parts, and some yellow caseous material, some of which may have broken down into irregular cavities filled with pus; and with this caseous material there may be, if it is a long-standing case, some calcareous material. The capsule of the gland is much thickened. Several adjacent glands may be thus affected, their capsules being greatly thickened, and the glands being adherent. When we meet with such a mass of enlarged and adherent glands we may have to inquire, "Is this malignant? Is this enlargement due to struma, or is the case one of carcinoma, or of lymphadenoma?" One aid in deciding the question would be the duration of the swelling; if it has existed for years, it is evidently not carcinoma, but struma.

What do we see on microscopic examination of a strumous gland? We see lymph-cells heaped up until they form a darkened opaque mass charged with small granules. Many have lost their nuclei, and are filled with fat droplets; they have undergone fatty or caseous degeneration, and in some portions of the mass there is not a trace of cell-structure. It is entirely broken up into granular matter. In another part the cells are very much out of shape—cells irregular from pressure and partial disintegration surround the caseous core of the gland. Outside this are clearer spaces—lymph-spaces—where the cells are not so numerous, and have not undergone so much change. As the lymphatic gland becomes swelled and packed with this large accumulation of cells which are rapidly dying, the vessels of the gland become pressed upon. Owing to this pressure the flow through the vessels is weakened, and they become dilated from passive congestion, and there is increasing tendency to inflammatory exudation. As the vessels are ever more and more compressed by the swelling, there is increasing tendency to inflammation and destruction. Such is a strumous process.

A strumous constitution produces phthisis. Scrofula and phthisis are inseparable. If I describe to you the microscopical appearances of a lung which has undergone strumous inflammation, you will have a fuller understanding of the strumous change, and I need not recur to this matter when I come to lecture on phthisis. We notice that the capillaries are enormously engorged, and in the tissue around the capillaries we see a large number of cells that look like lymph-cells and colourless blood-corpuscles. These bodies are similar, if not the same. The capillaries are buried in large quantities of these lymphoid corpuscles, which have undergone fatty degeneration; the alveoli and the bronchioles are filled with them. As the process advances the blood-vessels, air-tubes, and connective tissue disappear, and after a time the cells themselves are destroyed and broken up into a granular material. Why does this occur? The inflammatory material as it is formed destroys the natural elements of the tissue, and then, as the strumous deposit gets larger, and as the blood-vessels are pressed upon by the accumulating strumous material, the blood coagulates in them, and the nutritive fluid is cut off from the tissues, the inflammatory cells no longer have enough food, they undergo fatty degeneration, and die.

Such an inflammatory strumous deposit is known as a tubercle. In such accumulations we commonly find large irregular cells which have been called tubercle-corpuscles. These irregular corpuscles occurring in connection with caseous material are not confined to the lung, and though they are characteristic of, yet they are not limited to, tubercle. They are many times larger than an ordinary cell, contain many nuclei, and are charged with granular material.

If asked, "What is the tendency of tubercle?" say, "To loss of organisation." First we saw the natural organisation of the tissue replaced by the lowly organised inflammatory cells and the tubercle-corpuscles. The inflammatory material, as we saw, soon undergoes fatty and caseous degeneration, and a tubercle-corpuscle is no sooner formed than it begins to perish and lose its form. On microscopic examination, it is difficult to get the light through a strumous accumulation, owing to the loss of organised cell-structure and its replacement by tightly packed granules.

I told you that the vessels were compressed until thrombosis occurred; the red corpuscles also undergo degeneration, and the hæmoglobin they contain is transformed into pigment. Hence strumous tuberculous material is always pigmented in some degree, in the lung extremely so. Do not forget the plugging of the vessels in the lung as one condition that may prevent death by hæmoptysis in phthisis; for the same reason profuse or fatal hæmorrhage is not common with strumous changes elsewhere. Thickened fibroid tissue around strumous disease is very common, and in phthisical lungs it is notably common. It is an attempt to repair—an attempt to limit the morbid change.

Strumous disease of the testicle usually begins near the epididymis, towards the back of the organ. There may be simple strumous conditions of a catarrhal kind, with some amount of catarrh extending up to the ureter; and this catarrh may subside, or it may be repeated until there is a very swollen and firm testicle, containing much caseous material. This may break down into an abscess and discharge externally, and may go on discharging until the dead material is removed, and then the sinus may heal; or fibroid or caseous deposits, with atrophy, may remain.

We will next consider strumous disease of bone. This occurs in anæmic and badly nourished children, living under unhealthy conditions. In such, acute inflammation of the bone may rapidly kill by diffuse suppurative periostitis and ostitis, which we sometimes see involving the whole tibia and leading to death. At the post-mortem examination we find the periosteum has been stripped from the bone by the formation of pus beneath it. This acute condition is not however strumous. Strumous inflammation may attack a very small portion of bone, simply taking a form of ostitis corresponding to the early strumous catarrh we see in the larynx, the kidney, and other organs. If there is not a clear history of a blow, the onset of such inflammation denotes strumous weakness of the constitution. At first there is pain and tenderness, followed by swelling, which after a time becomes fluctuating. There is suppuration, with external discharge. On passing a probe dead bone is felt, and we know that there has been caries, or eating away, and necrosis, or death, of the bone. The process is very similar to that which we have described in the case of the other organs. As the inflammation proceeds, it destroys the natural structure of the bone, and dissolves the calcareous matter. If asked, "What is caries?" say, "A subacute inflammatory process of a strumous nature, which spreads and spreads and disorganises the bone, softening it down into a cavity, the edges of which look as if they had been eaten away. There is pus in the cavity, and, on examining the bone of which the wall is made up, we find that it is pigmented, and it may be studded with caseous matter." The origin of caries is always tuberculous caseous matter. The strumous change in bone may begin in one part and spread widely, or there may be a number of isolated centres.

On examining the blood of strumous patients, a diminished number of red corpuscles, and an excessive number of colourless ones, have been found.

What are the conditions which long experience teaches us have given rise to scrofulous mal-nutrition? First, inherited weakness in the tissues of the body. Second, unhealthy hygienic conditions; therefore scrofula is prevalent in large towns, with overcrowding, damp, confined, and bad air, and insufficient light, along with inadequate and improper feeding and clothing. By inadequate is meant, not according to the necessities of the body, which has to grow and develop to make healthy progress. What is meant by improper? That the food and the clothing are not in accordance with the properties and wants of the tissues and circulation. Immense difficulties are put upon the heart and respiration when the clothing is improper, and strumous patients especially suffer much from cold and heat. An enormous number of infants die under one year of age from diarrhœa and other mucous diseases, which are largely dependent on improper food and clothing. History has revealed that strength of national character is much dependent on proper food and clothing, and hence we should be careful to give food according to the work that has to come out of the body. Scrofulous children are mostly sensitive and too much "on the go," and so they require much food; and they require food that will absorb oxygen quickly, such as animal food, and plenty of water. Another cause of scrofula is excessive excitement, which hinders rhythmical action of heart and breathing. There is not one cause of scrofula, but many causal conditions combined are required to bring it about

LECTURE V.

RICKETS.

Rickets and scrofula have some intimate relations. Like scrofula, rickets gives us very instructive evidence that there are some unhealthy conditions which hinder the healthy progress of the blood and serum through the body; and in rickets there is a remarkable illustration that the nutrition of bones, the nutrition of mucous membranes, the action of the nervous system, and the rhythmical action of the windpipe must work harmoniously. That fact we have to bear in mind when we come to the treatment of rickets.

Years ago rickets was attributed to a deficiency of lime in the bones, and therefore lime-water and phosphates of lime were given, but failure was the result.

In the early stage of rickets one of the symptoms is excessive sweating, clearly showing that there is some failure in the government of the water of the body. Another early symptom is hyperæsthesia; the child dreads to be moved, and cries whenever it is lifted; these conditions show some unhealthy action of nervous tissue.

At this early stage we may meet with other disturbances in the nervous system, and the child may die quite suddenly. Dr. West says that in many cases of supposed overlaying, death is really due to laryngismus stridulus in connection with rickets. In every case of rickets we should inquire if there are any symptoms which point to an affection of the windpipe. For suddenly a child may be seen to fix its eyes, a look of alarm comes into its face, there is a passing shade of blueness, followed by pallor and probably lividity; then a croupy, crowing inspiration, and the spasm has gone. I say spasm, but it is uncertain what the actual condition is ; whether the cords are thrown into some rigid contraction, or whether they are partially paralysed, we do not know, but whichever way it is the air has great difficulty in getting down the trachea, and there is danger of suffocation. The child may die in the first attack, or the spasms may return many times for months. In the attacks of spasm we are on the look-out for convulsions ; if the spasms are severe there may be convulsions, ending in recovery or death.

If we are called to a case of croup, we should suspect rickets if the child is under two years of age. If we find the anterior fontanelle widely open, the ribs sinking in, if we find that the child has been sweating excessively for weeks past, we know that there is rickets, and that there is danger of sudden death. We cannot usually distinguish the posterior fontanelle, but the sutures are not properly closed up, and the forehead projects unduly, and the top of the skull is flattened on account of the retarded ossification. The head looks large in proportion to the face, and the question is asked, " Is there water on the brain ? "

How is it to be known from hydrocephalus ? Chronic hydrocephalus is a malformation which is present from the time of birth, whilst, on the other hand, rickets comes on in the latter half of the first year of life.

We must further notice that in rickets the extremities of the long bones are thickened, enlarged about the epiphyses. With the enlargement it is evident that the bones are softened; they cannot bear the ordinary pressure, and therefore many kinds of curvature come on. The clavicle and ribs are softened; the latter sink in on either side of the sternum, owing to the traction on them during inspiration, and, the sternum being thus pressed forward, there is produced the pigeon-breast malformation of rickets. The curvatures of the spine and of the extremities are increased, and the chest is so much thrown out of shape that the child may be unable to sit up, being bent like an old man, and dreads to be moved because its breathing is so short. The shortness of breath is due to various conditions ; one is that the ribs sink in and press upon the lungs during inspiration, squeezing the anterior parts of the lungs forward underneath the cartilages, and thus their anterior edges are rendered emphysematous. Secondly, bronchitis is very common with rickets. Whenever you meet with a child under two years of age suffering from bronchitis, examine for rickets. Thirdly, the circulation is feeble; the muscles of respiration, and other muscles, including the heart, are weak. Fourthly, there is disordered rhythmical action in the vocal cords, with more or less spasm.

The bronchitis of rickets is suffocative in its tendency, being accompanied by a large amount of exudation, and for this reason you are warned not to give antimony and alkalies. In rickety children the bronchitis recurs and recurs, and this condition may last for many months; but as the mal-nutrition of the rickets disappears the patients lose their bronchitis; there is wonderful recovering power in rickets, therefore hold out hope. But to assist this recovering power you must go on treating the rickets, which is the cause of the bronchitis. All this time there is much sweating of the skin, and sweating of the mucous membranes, catarrhal diarrhœa, which may alternate with bronchitis; skin affections are also often present, as eczema, lichen urticatus, or impetigo; but these various morbid conditions disappear under the treatment for rickets—cod-liver oil and iron.

The seat of the morbid changes in the long bones is in the ossifying areas on either side of the epiphysial cartilages; there is some irritable weakness in the cartilage cells, which swell and multiply excessively, thus leading to the thickening of which I spoke above. The alveoli around the cartilage cells are much enlarged. These alveoli are really serous spaces connected with the medullary cavities in the shafts of the long bones. There is congestion of the vessels which come from the medullary cavities and spread into the cartilages, and hence some regard this morbid change as a kind of subacute inflammation of cartilage and bone. There is much thickening of cellular material in the immediate neighbourhood of the vessels, but the hyaline matrix of the cartilage is much diminished, being riddled by the enlarged lacunar spaces. As this occurs, the bony growth in the hyaline substance between the rows of cartilage cells is lessened and removed, and the cartilage softened.

The conditions of rickets are due to inherited weakness, to insufficient and improper feeding, to being kept too much indoors, and not enough in association with the open air. If you keep indoors for a week, see how nervous you will get. Outdoor work and healthy surroundings combine to develop healthy vis nervosa.

LECTURE VI.

SMALL-POX.

The initial symptoms of small-pox are characteristic, and are common both to mild and severe cases; they are, pain in the back, pain in the head, and vomiting. From these initial symptoms we cannot tell whether the attack is going to be mild or severe; to ascertain this we have to wait for a day or two. If the attack is about to be severe, we are soon impressed by the increasing prostration, and by the onset of severe inflammation of the skin; the face becomes red, much swelled, and œdematous. At this stage it might be mistaken for erysipelas, and you must remember that the variolous eruption may be preceded by a measly or scarlet rash; or at the time when papules are appearing on the face such a rash may be seen in the groins or the flexures of the knees.

When it is a mild case of small-pox (by mild I mean not confluent), we notice small red areas of congestion on the face on the third day, the next day they become somewhat papular, on the succeeding days vesicular, and then the contents of the vesicles become yellowish towards the seventh or eighth day. An eruption of this kind is scattered over the body. During this time the temperature has been raised, but it falls towards the eighth day—from the sixth to the eighth day. In these mild cases the temperature never rises very high, and on the ninth, or at latest the tenth, day it is normal. There is no secondary fever, the contents of the vesicles dry up and are cast off in a very few days, and the patient is soon well. Now that is the common course of modified small-pox. Patients suffering from it sometimes walk about with very little disturbance of their general health. I may recall to you that it is sometimes difficult to distinguish these mild cases from chicken-pox because the eruption in both is vesicular, and they both pass through their course in a few days with very little feverish disturbance, and, if you are in any doubt, do not attempt to be positive.

In confluent small-pox the suffering and the signs are very different. The skin of the face, neck, arms, chest, and back is observed to be much congested and swelled, and we see papules studded as thickly as they can be placed; the skin of the abdomen and lower extremities is similarly affected. In the next two days the papules become much raised, and pass into vesicles, so that the skin is entirely covered with vesicles. In some cases (formerly termed pearl-pox) it is surprising to see how large are the vesicles and how watery their contents. The vesicles become opaque after the eighth day. The temperature in these cases from the first to the fourth day goes on increasing; after the appearance of the eruption it falls somewhat, and goes on falling until by the eighth day it may be down to 100° or 99°, very little above normal. On the ninth day it begins to rise again, and goes on rising until the twelfth day; this is the period of suppuration, when the serous contents of the vesicles is being converted into pus, and it is at this time that confluent small-pox presents its most hideous appearance. The whole body is covered with pustules which have pressed against and run into each other; some of them have burst and pus is oozing from them, while the skin is smarting and burning, so that rest is impossible. When the eruption is coming out on the face the throat becomes congested, and we see small grey patches on the soft palate and tonsils, suggesting that there is a vesicular eruption on the throat similar to that on the skin; but it does not pass into a pustular condition in a similar manner; or rather, to put it more accurately, the vesication and suppuration on the soft palate and fauces are much less noticeable.

As the irritation in the skin increases, we have to call to mind that the skin is a great excitor and regulator of breathing, and we may therefore predict that whenever the skin is greatly inflamed, danger may come in the breathing organs. It is so in burns, in erysipelas, in small-pox, in pemphigus, and other skin affections. There are many illustrations to show that in proportion to the degree of irritation of the skin there is risk to life from failure of the respiratory organs. In confluent small-pox, as the irritation in the skin increases the breathing becomes quickened, and as the breathing becomes more disturbed the patient becomes extremely restless, sleepless, and delirium comes on. We notice that the diaphragm begins to act excessively, and we recognise that the lungs are becoming congested; it acts with abnormal energy to get air into and out of the lungs, and so we measure the degree of danger by the action of the diaphragm. It is necessary to take this guidance, for we cannot and ought not to make the patient sit up in bed to examine the chest. As this stage of secondary fever and suppuration progresses and the skin irritation increases, we notice that the epigastrium sinks in much with each inspiration, and we find that the pulse-tension is increasing: these signs are a prelude to suffocation, and so in fatal cases these unfavourable symptoms become more and more marked until about the twelfth or thirteenth day; then extreme restlessness, wakefulness, and delirium are followed by increasing stupor, passing into coma; a cold sweat appears on the skin, the temperature falls, the pulse loses its fulness, and we know that the patient is sinking.

What do we find in such cases on making a post-mortem examination? Take note of that, for I have been surprised to find how little some students know of it when questioned. We find the larynx and trachea most extremely congested, and in some degree the larger bronchi also—that is one of the most striking features at a post-mortem examination of small-pox. The mucous membrane of the upper air-passages is swelled, granular, and of a purple colour, often about the colour of port-wine, and there are scattered hæmorrhages in it. The bronchial tubes and the lungs are found to be extremely congested, and in parts black from extravasated blood. The lungs weigh very heavy, for, in addition to the excessive distension of the vessels, œdema of portions of the lungs, and patches of extravasated blood like to pulmonary apoplexy, there are patches of pneumonic consolidation, both red and grey hepatisation, and the bronchial tubes contain more or less muco-purulent matter. The right side of the heart is extremely distended by accumulated blood, and there is extreme venous congestion of the brain and of the abdominal organs.

The post-mortem examination and the clinical experience therefore manifest that in confluent small-pox danger to life, and death, are due to failure of the respiratory organs: so when we are estimating the severity of an attack of small-pox, what we have to consider is the degree of irritation of the skin, and whether the tissues have undergone much chronic degenerative change. At middle age, as you know, vesicular emphysema of the lungs is common, and such emphysematous lungs, which being weak antecedent to the small-pox, are very commonly attacked by broncho-pneumonia, which ends fatally.

If the patient is going to recover, more refreshing sleep returns, the breathing becomes much easier, and the fever begins to decline about the twelfth or thirteenth day; the pustules become very yellow, and some of them burst and their contents escape and dry up; in others the contents are absorbed and the pustules dry up without bursting. After they are dry and shrivelled the epidermal remains are cast off. In modified small-pox the inflammation does not extend much below the epidermis, and there is therefore very little subsequent scarring; but the inflammatory processes in confluent small-pox extend deeply into the subcutaneous cellular tissue, and destroy the pigment layer, so that white scars are left.

The nutrition of the skin and the general circulation and the breathing are left weak for two or three, or it may be for many, weeks after the attack, and, when the patient seems to be gradually recovering, we may be surprised to find that suddenly a rigor comes on, and the temperature may rise to 105° or higher, and on examining the patient we may find no evidence of pneumonia, or other acute inflammation. This may make us doubt if there has been a rigor, but careful inquiry leaves no doubt of that, and in the course of the next two or three days, during which the temperature may continue raised, we may find that this severe disturbance has been due to a local inflammation of the skin, taking the form of a boil. Such rigors may be repeated three or four times during the next few weeks, pustules and boils continuing to form in the skin, so that the convalescence is extended through several months. Now, throughout this period, the lungs being weak, broncho-pneumonia may supervene, and may end in phthisis; or there may be nephritis, or other inflammatory disease.

We have to remember that when the breathing has been much disturbed, the brain and mind have also been much disturbed, and the patient may in the convalescent stage become insane; he is observed to be increasingly restless, silent, morose, sleepless, until there is no doubt of the mental aberration.

Here let me remark that we must not keep a patient with small-pox in a confined atmosphere. If you turn to the early editions of Watson's "Practice of Physic," you will find that he says that it was a great advance in the usual treatment of small-pox when less clothing and more air were suggested. The windows of the room should be opened from time to time, and the body should be sponged and some oil applied to soothe the skin. Experience has shown that many more recover when the rooms are well ventilated; we have as far as possible to aid the breathing until the disease subsides.

I pass on to speak of malignant small-pox, which once seen cannot easily be forgotten. Two varieties may be described. In one, the skin of the face and neck on the third day becomes extremely red, congested, and œdematous, and looks like exceedingly severe erysipelas. Then bleeding from the nose comes on, the gums bleed, blood is coughed up, blood is passed with the urine, blood is extravasated into the skin, and these hæmorrhages extend rapidly all over the surface of the body, and the patient dies before there is time for suppuration, or even before there is time for papules or vesicles to form distinctly.

In other cases the redness of the skin is replaced by an extreme purple. The skin over the whole body is hard, œdematous, purple, and is everywhere studded with papules and vesicles, which are so thickly placed that they are indistinct; these patients are conscious and remarkably calm, and complain very little, but the pulse gets smaller and smaller, the eyes are kept open, there is extreme wakefulness, for collapse is setting in, the breathing becomes more and more labouring until death. The peculiar indifference with this most fatal suffering rivets our attention. I know nothing in disease that has impressed me more than the fearful alteration of the skin and the chilling indifference in the patient's aspect that I have observed in these malignant cases. They end fatally on or before the fifth day. The morbid appearances we find at the post-mortem examination are similar to those found in cases of confluent small-pox.

Here let me mention to you that we might on post-mortem examination hastily mistake this condition for purpura. I was asked once to make a post-mortem examination on the body of a man who had died mysteriously. I could only find evidence of purpura, and had I been guided merely by my examination, I should have had to say that the cause of death was purpura; but the appearances were so curiously like and unlike purpura, not altogether like other cases of purpura on which I had made post-mortem examinations, and the blood-extravasations in the skin were so large, that I thought there had been some morbid change more than the post-mortem revealed to me. On inquiry, I found that the clinical history tended to show that most probably the case had been one of malignant small-pox, in which death had occurred from hæmorrhage before there was time for the eruption to come out; it was a most extremely malignant case. Bearing upon this I may here tell you that a person may die of some malignant infectious disease before there is time for the crucial distinctive signs to appear, as in cholera, dysentery, or, it may be, in malignant scarlet fever or small-pox.

LECTURE VII.

CHOLERA AND DIARRHŒA.

WHAT are the distinguishing features between cholera and diarrhœa? During an epidemic of cholera we have hourly to face this question, and almost every summer attention is drawn to an exceptionally severe case of diarrhœa in adults, in which there is collapse, and the question arises, "Is it choleraic diarrhœa or English cholera so termed, or must it be regarded as an instance of Asiatic cholera?"

When I was physician to the Cholera Hospital I used frequently to hear the resident medical officer say, "This patient has not got cholera, but choleraic diarrhœa." "What makes you say that so dogmatically?" I often asked him; and he would reply, "Oh, he has been having colic and diarrhœa for the last three or four days, but when a man has cholera he has painless diarrhœa, and cramps in the calves of his legs." That experience was repeated so often that we took it as a rough-and-ready guide.

Choleraic diarrhœa is distinguished from Asiatic cholera by the colicky pains in the belly recurring for hours or days with little or no cramp in the legs, and by the evacuations being watery, coloured, fæcal, and containing sometimes a good deal of bile. The distinction lies in this, that in diarrhœa and in the several varieties of choleraic diarrhœa the patients do not pass rice-water stools, and we do not see the characteristic rapidly repeated vomiting and purging which quickly lead to collapse in Asiatic cholera.

During the months of August and September, when the air is very hot and very moist, and there is a great deal of decomposition going on in organic materials, and when summer diarrhœa is very prevalent, we now and then see a case in which purging and vomiting have been so rapidly repeated that the patient has passed into a condition of partial or complete collapse, and the evacuations from the bowel are colourless and exceedingly watery, like thin rice-water, with now and then a little gelatinous material in them; then we have to ask ourselves, "Is this Asiatic cholera, and is there going to be an outbreak of the epidemic form?" For years I have seen somewhat similar cases nearly every summer, and on several occasions I have been called in consultation to decide as to the nature of the attack, and especially with a view to prognosis and treatment. They are cases of English cholera. The collapse was very similar to that of Asiatic cholera, but not so extremely marked; the skin was cold, there was more or less lividity, the eyes were sunken; but there was this distinguishing feature in every one of the cases, that they were not completely pulseless; the pulse was exceedingly weak, so that I could only just feel it, but it could be decidedly felt, and so I was led to say to the medical attendant, "I think that the patient will recover." They generally do recover, but I have known a few such patients die in the hospital, and when I made the post-mortem examination I found no distinct rice-water-like contents in their intestine, nor the extreme congestion of the reactive stage, as is seen in cases of Asiatic cholera.

I have, however, at long intervals seen cases that I could not distinguish from Asiatic cholera, but they were isolated cases occurring in the summer, and the disease did not spread and attack other persons, as in an epidemic of Asiatic cholera. It is considered that these are sporadic cases of Asiatic cholera which may be met with in the summer, just as a sporadic case of small-pox may be seen in the absence of an epidemic.

The rice-water evacuations of cholera denote that all fæcal matter has been swept out of the bowel. You might ask, "How do you know that?" Because I have made post-mortem examinations in many cases of Asiatic cholera, and when death has occurred in the stage of collapse, I have found that the bowel contained no fæcal-stained fluid from one end to the other. During the epidemic of 1866 I opened body after body and found that the intestine contained nothing but the rice-water fluid.

In general practice you will meet with many cases of children dying from summer diarrhœa. The attack begins with vomiting and purging, and exceedingly watery fæcal-coloured evacuations are passed from the bowel, and the motions may be so frequent and so large that collapse sets in in a few hours. The post-mortem examination in a case of diarrhœa reveals very little. We find the intestine anæmic, and its mucous surface has an exceedingly granular appearance, especially in the ileum, showing that the epithelium has to a great extent been shed. Such are the morbid appearances if, as is most common, the child dies in the stage of collapse. If death occurs in the stage of reaction, when the evacuations have become more scanty and consist of bile mixed with mucus, we commonly find much congestion in the lower part of the ileum, and there may be superficial ulceration of the solitary glands or of Peyer's patches.

Before leaving these cases of diarrhœa in infants it will assist you if I mention that the extreme exhaustion resulting from diarrhœa may be associated with cerebral failure to such a degree that the symptoms have been compared to those of acute hydrocephalus, and this condition of collapse from diarrhœa in infants has been called spurious hydrocephalus. The infant is seen lying extremely restless, or taking no notice, seeming insensible, with sunken eyes, labouring breathing, nearly pulseless, and every now and then uttering a piercing cry—very similar to what is witnessed in acute hydrocephalus. But to avoid mistake it is only necessary to inquire of the antecedents, to ask if the condition has resulted from diarrhœa.

The initial diarrhœa of Asiatic cholera is a watery fæcal evacuation, like that of ordinary diarrhœa, and is usually painless; but after a few days, it may be after a few hours or minutes, the patient is seized with severe and rapidly repeated vomiting, and brings up a quantity of watery-looking fluid. All fæcal matter is soon swept out of the bowel, and the evacuations consist of a serous fluid. This fluid has pieces of mucus floating in it: such is the characteristic stool of Asiatic cholera, and from its resemblance to the water in which rice has been boiled it is commonly known as the rice-water evacuation. With this there are severe cramps in the calves of the legs and in the belly, and sometimes also in the arms, and owing to the cramps the body is violently distorted; the temperature of the periphery falls, it may be to 95° or even lower (the temperature thus extremely low in the axilla or mouth may be extremely high in the rectum during this stage of collapse), the skin becomes pale and more or less livid, the breathing is labouring, the pulse becomes smaller and smaller until it cannot be felt, the eyes are sunken, the tongue is cold, the breath is cold, the voice is reduced to a whisper. We can estimate the degree of collapse by the tone of the voice. While lying in that condition the patients are seen breathing slowly and laboriously, their eyes widely open, and conscious, but their restlessness is most extreme, and they keep on asking, "Can you give me more air?" And yet on listening to the chest the breath sounds denote that air is entering and escaping abundantly into and from the bronchial tubes; I used often to listen and thus hear the air entering freely into the chest, and passing out again freely, and subsequently at the post-mortem examination I have found that the bronchial tubes were clear from any foreign accumulations of serum, mucus, or pus, to cause obstruction to breathing, and yet these patients suffered extremely from want of air, from *Lufthunger*. How then was it that the breathing was fatally failing? Well, I can in some degree answer that question: it was because the normal exchange between the water of the blood and the air in the alveoli had become impossible—by the vomiting and purging the water had been enormously drained away from the lungs and other organs and tissues, and therefore the *besoin de respirer* was agonising. But it was wonderful to see the change when we ran water at the temperature of 100° into the blood. A minute quantity of chloride of sodium was added to the water to simulate serum, a vein was opened, and the water allowed to run into it from a vessel suspended a few feet above the patient's arm. As the water passed into the circulation it was amazing to see the breathing relieved, the pulse return, and the symptoms of collapse disappear.

A woman was lying in complete collapse in the Cholera

Hospital. I waited until there was no pulse and no sign of breathing, and all sign of life had ceased, then I let the water run into a vein of the arm. The breathing returned, the pulse returned, warmth and colour returned, the patient opened her eyes and spoke, but unfortunately the improvement did not continue. We gave these injections in many cases and the patients rallied wonderfully, but it was only for a few minutes or for an hour or two, and then they died.

What do we find on post-mortem examination in these cases of cholera in which death occurs in the stage of collapse? We find the belly sunken, the eyes extremely sunken, the cheeks sunken, the hands looking as if they had been soaked in water for days—the "washerwoman's hand." Evidently the cellular tissues have been largely drained of their water. We used often to estimate the degree of purging by looking at the shrunken skin of the hands. On opening the abdomen the intestines are seen contracted, in many cases empty and lying packed together towards the spine, or filled with rice-water-like fluid, and we find the mucous membrane of the bowel exceedingly anæmic and granular, particularly so in the jejunum and ileum. The lungs weigh much less than normal, about twenty ounces instead of forty; when cut into they look livid and exceedingly dry, so that we can scarcely squeeze any fluid out of them; the bronchial tubes are free from accumulations, containing very little mucus, but showing some venous congestion; the kidneys, spleen, heart, and other organs all weigh much less than normal because the water has been largely drained away from the body.

In the early period of a cholera epidemic the disease is witnessed in its most virulent form. Of those who pass into collapse very few recover. Speaking from my own experience, I was impressed that not ten per cent. of those in whom the collapse was so extreme that we could not feel the pulse, recovered. Later in the epidemic, when it was showing signs of subsiding, many more recovered, as many as fifty per cent. Moreover, at the outset of the epidemic its virulence was evidenced by the extremely rapid course of the disease: persons were seen in whom the distinctive vomiting and purging had commenced an hour or so before, they then showed no sign of collapse, but in another hour or two the collapse was fatally marked. In one instance (cholera sicca, so termed) the patient was seen walking along the pavement when he suddenly dropped, and, although it was near the London Hospital, before he could be brought in he died. I found the lower part of the large intestine filled with solid fæces, and the remainder of the colon and the small intestine filled with the characteristic rice-water fluid : the lungs in this case were not dry, but œdematous. Death had evidently occurred before the usual results of collapse could set in.

The post-mortem examinations showed in many no sign of reaction—the mucous surfaces anæmic, rice-water-like contents in the intestine, the lungs comparatively dry and weighing much less than normal, and the left ventricle of the heart firmly contracted—but in many others the mucous membrane of the ileum was more or less congested and bile-stained, showing that reaction had set in ; and the lungs also weighed heavier and contained more blood-stained fluid.

Some of those who were in complete collapse gradually passed into reaction, the lividity disappeared, the pulse returned, the breathing became much easier, the restlessness ceased, and they passed into sleep. Some of the younger subjects, boys and girls, slept for short intervals for a day or two, and then sat up in bed and said, "Please, sir, may I have a red herring?" or, "May I have some bread-and-butter?" and rapidly recovered their strength from that time. But such rapid recovery was rarely seen in adults; it was slower.

In others the reaction set in, but they died before it was much advanced. The lividity disappeared and the cheeks became flushed, the eyes were staring and expressing excitement, delirium came on, the pulse was somewhat full but very soft, the breathing was quickened, and then the pulse again became weaker and weaker, the breathing was again labouring, and they died.

We studied how we might promote and expedite reaction. We gave them water to drink abundantly, but they vomited it. We put them into hot baths, for we considered that they wanted heat and water; some of them were wrapped in blankets wrung out of hot water, and two beds were used, the patient being moved from one to the other as the blankets got cold. With such treatment the pulse returned, the colour returned, the warmth returned, but they became violently delirious. It did no permanent good. In the same way the injection of water into the blood helped them, but it was only for a very short time.

We had to ask ourselves how it was that the improvement did not continue. I will try in some degree to answer that question, and let me first say that when the circulation of the blood is completely arrested, if only for a few minutes, the red blood-corpuscles, it would seem, largely perish, and although the circulation may again be restored, it will not continue for more than a few hours—the blood cannot be retained in the vessels. This I have witnessed in cases of drowning, in which a man has been immersed for a few minutes, and apparently dead when taken out, but with subsequent treatment the pulse and breathing have returned, to cease finally after some hours. Similarly, I think in cholera collapse the blood undergoes destructive changes; and so in some cases of extremest collapse, though we may revive them for a time by running water into the blood-vessels, yet the changes in the blood have been too destructive for recovery to be possible—respiration cannot continue. That fatal changes have taken place in the blood and vessels is manifested by the bloody evacuations from the bowel that are often passed at the beginning of reaction, when many of the patients die.

In observing the phenomena of reaction we find that the serous discharges from the bowel cease, and that the evacuations become more bile-stained, and as the reaction becomes more pronounced, more and more bile appears in the intestine; that is a sure sign that the patient has passed out of collapse. Then the surface temperature rises, he becomes drowsy and somewhat delirious, the pulse returns, the skin begins to put on a congested appearance, the cheeks become flushed, the eyes bright, breathing becomes freer, the temperature may rise to 101° or more, and the sufferer is then in the stage of reaction. In other cases, such as I have alluded to, in which the blood undergoes destructive changes, so that respiration cannot continue, and bloody evacuations come from the bowel, the patients passed in some degree into reaction, and then they began to pass motions which looked like red jelly with little red lumps in them, the pallor of the skin disappeared and was replaced by a livid red, most marked in the face, the hands and the feet, the pulse could be felt for a few hours, the breathing remained difficult, wakefulness continued, and there were repeated evacuations from the bowel consisting of blood-stained fluid with pieces of bloody mucus floating in it. This condition continued for some hours, and then the pulse became smaller until it could not be felt, a cold sweat came on, and death soon followed.

Many other cases went further into reaction, but passed into a condition very similar in appearance to typhoid fever; the tongue dry and parched, with sordes on the lips and teeth, and there was continued fever, delirium, quickened breathing, and rapid weak pulse, and in many cases a fatal issue.

There was another symptom I must speak about, as it attracted much attention: I refer to suppression of urine in cholera. During the collapse which is consequent on the severe purging the secretion of urine ceases; as reaction comes on the urine becomes more abundant, but in these typhoid conditions it commonly remained very scanty, and commonly contained albumen. Similar phenomena are not limited to cholera, they occur in collapse from other causes, but in this form of reaction we often noticed more or less stupor, and sometimes convulsive seizures, and such symptoms as have been attributed to failure of kidney function, or uræmic poisoning consequent on the suppression of urine. I was soon led to consider that this view is erroneous. For, as said, there is like suppression of urine in collapse from other causes; for instance, after protracted vomiting of large quantities of liquid in a case of strangulated hernia, suppression of urine is seen as the circulation fails. By clinical experience and by post-mortem examination, finding in such cases that the kidneys only showed signs of venous congestion, I came to the conclusion that the stupor and other symptoms of failing cerebro-spinal power observed in cholera are due, not to uræmia, but to the feeble circulation, to the venous congestion of the nerve-centres, and to the impaired quality of the blood flowing through them. I do not think that the chief cause in that impairment of the quality of the blood is failure of kidney function.

Let me now finish what I have to say to you about reaction, by detailing to you the morbid appearances observed on making a post-mortem examination. The appearances are the reverse of what is seen after death from collapse. In the place of anæmic changes in the mucous membranes there is extreme congestion. There is intense congestion of the intestine, particularly in the ileum, the mucous surface has a deep red appearance like the colour of port-wine or claret, it is more or less bile-stained, and occasionally we meet with some superficial ulceration. The lungs weigh much heavier than normal, are congested, and there are patches of consolidation from recent pneumonia, and perhaps we find pus in the bronchial tubes. The kidneys, spleen, and other organs are similarly much congested, and the left ventricle of the heart not contracted and empty, as in collapse, but flaccid and full of blood.

I now pass on to speak of the causal conditions of cholera. I have already referred to epidemic Asiatic cholera and to cases of sporadic cholera, and I have admitted that when cholera is not epidemic in this country cases may be seen so similar to Asiatic cholera (differing, however, in this, that the characteristic rice-water evacuations are not present), that they have been regarded as sporadic cases of Asiatic cholera, and as affording evidence that the conditions for the production of cholera are more or less present in this country every summer, and that a great increase in the virulence of these conditions might lead to the outbreak of an epidemic. The logical bearing of that reasoning is this, that if the summer were such as to produce poisonous conditions by a great amount of decomposition, and a wave of contagion spread into the country, then there would be an epidemic of cholera. What do we mean by a wave of contagion? History gives a clear answer to that question. It is notorious that in the great epidemics of cholera the disease has spread from India in a great wave, either along the Persian Gulf, and by trade with the East has been brought to Southampton or some other port, or else it has passed across Asia into Russia, and has thence invaded England through Sunderland. It has been recorded that cholera does not occur in India in the hottest seasons nor in the wettest seasons. Heavy and continued rains seem to wash away the noxious conditions, and an extreme degree of heat seems to dry them up. The conditions most favourable for an extreme degree of chemical decomposition in organic matters are comparatively stagnant air, moisture, and a moderate amount of heat. Cholera occurs with most severity in places in which there is great accumulation of organic refuse of animal origin, it follows the tracks of pilgrimage, and it follows armies, where the earth and the air seem to get saturated with the products of decomposition of organic matter. We must not overlook the fact that when once the epidemic has taken a severe form, cold does not stop its spread; one of the most severe of recorded epidemics occurred in Russia in the winter. Has cold influence in checking the spread of other epidemic diseases? Small-pox does not occur chiefly in summer or autumn, the wave of a small-pox epidemic begins usually in November, and declines towards May. It is the same to some extent with typhus fever. What are the conditions which favour the spread of these diseases? Comparatively confined air, a moderate amount of moisture, and a moderate amount of heat. We get these conditions in close unhealthy dwellings in winter, and when cholera or small-pox gets into such unhealthy houses, they spread rapidly.

Is the contagion of cholera carried by any means that we can handle? It is admitted that it may be transmitted by articles of commerce, and by clothing; it has been observed that in cholera epidemics the people who wash the clothes of cholera patients are especially apt to be attacked by the disease. Another vehicle for conveying the poison of cholera is water. Here I will admit to you that I find it impossible to understand how a potful of cholera fæces thrown into a river can be a fruitful source of contagion; the power of oxidation in running water is so great. I used many years ago to watch people drawing filthy water out of the Thames Pool, and putting it into their water-butts; in the course of a week or two that water was bright, clear, and pure. If there is that wonderful disinfecting process going on in water, why does it not destroy the poison in the cholera stools? But I am quite willing to accept the facts. In 1866 the area supplied by the East London Water Company suffered most severely from the cholera, and the river Lea, from which their water was drawn, was at that time in a most filthy condition. In the same year it was noticed that the people drawing their water from particular wells were affected more than others by cholera. But we must look at the question broadly; to say that a single cholera stool thrown into the Thames can lead to a cholera epidemic in London seems to me to be a *reductio ad absurdum.*

You will be asked, "Do you believe that cholera is infectious?" Answer that question simply, "I believe that people are infected with cholera, that something passes into them and infects them, and that this infective poison may be conveyed in several ways, by water, food, or clothing."

How is it, that whilst there are these poisonous conditions existing, some persons are attacked and others not, whether it is typhoid fever, cholera, or dysentery? This is a question to which much research must be devoted to find the answer. In cholera it is said to be the outcome of the determining action of the cholera bacillus. But it has not yet been made clear how far such bacillus is an indispensable causal condition, and how far it is a concomitant in a series of consecutive causal conditions, the sum of which might be spoken of as the cause.

Our erroneous conclusions in etiology are obscuring the results of our limited observations—a definite conclusion can never be scientific and have application to the living organism unless it embraces a series of consecutive changes. . . . .

LECTURE VIII.

DYSENTERY.

By dysentery we understand inflammation and ulceration of the colon and rectum. The disease is always most marked in the lowermost part of the bowel, where venous congestion is most apt to occur. A dysenteric inflammation may be excited by inflammation occurring in the neighbourhood of the colon; owing to perimetritis, for instance, an abscess may form, and burst into the rectum; this may excite much inflammation and ulceration of the bowel, spreading upwards and downwards from the opening of the abscess. There are bowel symptoms similar to those of dysentery, and the condition may be attended by more agonising suffering than is witnessed in dysentery. This condition, however, is not what we ordinarily think of when we speak of dysentery.

My experience in the post-mortem room has impressed me that there are three principal forms of dysentery.

I. Dysenteric inflammation and ulceration may result from long-continued venous congestion of the bowel, leading to œdematous swelling of the mucous membrane of the colon and rectum more particularly, followed by catarrhal inflammation and repeated ulceration. On post-mortem examination in a case of this nature we find that the mucous membrane is extremely venously congested, and of a purple colour, and the membrane is much swelled and granular, and a large area of it is superficially ulcerated. This form of catarrhal dysentery is liable to come on in diseases in which there is chronic venous congestion of the bowel—for instance, in cases of heart-disease, vesicular emphysema of the lungs, and empyema of long-standing; and for this reason it is advisable in such diseases to examine the motions from time to time, especially if you are told that they contain much mucus.

II. The second form of dysentery occurs in persons who have lived in close, confined places, and therefore it occurs in connection with overcrowding and bad ventilation. Sailors who are huddled together in unhealthy forecastles on board ship are liable to it. I have been accustomed to speak of this variety as arising from decomposing animal organic matters. On post-mortem examination we find the transverse veins of the large intestine very full; we see bands of congestion passing transversely across the mucous membrane, and the membrane in these congested areas is much swelled by exudation, and is also granular and ulcerated. There may be many such bands and many such centres of ulceration; further, the ulceration spreads until the mucous membrane is almost entirely destroyed by contiguous masses of exudation and ulceration. Both this form of dysenteric inflammation and ulceration and that last described, may involve the lower part of the small intestine in addition to the large intestine.

III. The third form has been described by some as "small-pox of the large intestine." We are impressed on making a post-mortem examination by the appearance of countless numbers of little projections, about the size of peas and of a greyish colour, studded all over the inner surface of the colon and rectum. These are evidently produced by exudation into the solitary glands. Some of these projections soften down in the centre by a process resembling that which occurs in typhoid fever: there is a crumbling away of the deposit which leads to the formation of an ulcer; these ulcers spread and spread until their edges run into one another, and so the mucous membrane is destroyed. It is usual to find in the upper part of the colon these characteristic deposits in the solitary glands; and as we pass down the colon the masses are seen softened and pallid in the centre, and the nearer we approach the rectum more and more ulceration is seen, and we observe that the entire mucous surface of the sigmoid flexure and rectum is completely destroyed by ulceration. This is the form of dysentery in which we meet with abscess of the liver—malarial dysentery. The abscess of the liver may either precede or follow the dysenteric inflammation. The illness may begin with malarial fever of the ordinary type, then dysentery sets in, and is followed by abscess of the liver.

The morbid appearances of these two varieties of dysentery are also described by Aitken in his "System of Medicine," but he does not attempt to trace any difference in their origin. Thus far I may say, that where I have found the typical deposits in the solitary glands there was a history of exposure to malarial infection, and, as said, abscess in the liver was met with; whereas in the other variety we could not trace any such exposure, but we found that the sufferers had been living in close confined places in which decomposing animal matters would tend to accumulate and to poison—hence, for convenience, I have spoken of one variety as malarious and the other as miasmatic dysentery.

More frequently death does not occur in dysentery until the disease is far advanced and the primary changes cannot be traced. What are the appearances of the mucous membrane in the later stages of dysentery? We find that the normal mucous membrane has entirely disappeared; the smooth translucent velvety appearance has gone. We see numerous little projections of a deep red colour, varying in size from that of a split pea up to half an inch in diameter; their colour is like that of red-currant jelly; they are extremely vascular, and their appearance suggests to us that they would be likely to bleed freely. Around and between these we notice a grey granular appearance; that is the muscular coat of the bowel covered with inflammatory material. The whole of the colon and rectum may present this appearance. The red projections are portions of the mucous membrane that have escaped complete destruction, but are most extremely congested, and contain small hæmorrhagic extravasations; elsewhere the mucous membrane has been entirely destroyed. We often find the bowel much contracted and empty, giving evidence of great irritation until all fæcal matter was expelled. The wall of the colon looks thicker than normal, but I think that this appearance is mostly due to the contraction; I have never been able to satisfy myself that there is true hypertrophy of the muscular coat.

We have further to inquire what are the appearances when there has been old dysentery, followed by recovery, and subsequently more recent dysenteric inflammation which has killed the patient. You should pay particular attention to this, for it is a common occurrence. When a man has once had dysentery the inflammation in the colon is apt to recur. In some cases there have been several attacks of this kind, until the most extreme exhaustion, emaciation, and pigmentation of the skin, have resulted therefrom, and from that cause I have seen patients, generally sailors, brought to the hospital nearly pulseless, the voice, as in cholera, extremely feeble, the temperature lower than normal, the eyes much sunken, the cheeks hollow, the skin dry and shrunken and almost black from pigmentation. Yet with rest, warmth, and medical attention, some have recovered wonderfully well, but many such cases ended fatally. In such cases we find a white milky fibroid appearance over a large portion of the mucous surface of the colon and rectum, and we know from that, that there has been old inflammation, and doubtless ulceration, which has left fibroid thickening and pigmentation; in addition to this we see patches of recent inflammation and ulceration such as I have above described.

I pass on to speak of the suffering in dysentery. The disease may come on very acutely with fever, which may be either of an intermittent or of a continued type. It is important to remember that in this country also dysentery may occur in children as an acute febrile attack, ending fatally in a few weeks. The constitutional symptoms are soon followed by a liquid fæcal discharge, like that of ordinary diarrhœa. The evacuations succeed one another at short intervals until all the contents of the bowel are swept out. In acute attacks this is followed by copious milky serous evacuations. In rare cases the patient may die of collapse at this stage—rare, that is to say, in this country; but it is recorded that in tropical countries patients sometimes die of collapse from dysentery before there has been a single evacuation. I have told you that we sometimes see the same thing in cases of cholera. If the attack is less acute the fæcal evacuations are immediately followed by evacuations of mucus and blood. We may in this disease, as with inflammations in general, estimate the severity of the attack by the amount of hæmorrhage. As the early acute inflammatory condition subsides, but while the ulceration continues, we find that the patient passes much mucus streaked with blood, and there is the characteristic tenesmus of dysentery, the desire of the patient to sit on the stool and strain; he strains much, and yet he passes nothing but a little blood-stained mucus at each evacuation. If you learn that a patient is frequently going to stool, and that he passes merely a little mucus, you know then that the case is not one of diarrhœa, but of dysentery, and that the reason that there is so much straining is because there is a great deal of ulceration immediately above the sphincter ani, and a very little mucus accumulated at this spot will cause great irritation until it is evacuated.

In bringing this lecture to a close it will aid you if I additionally mention that in cases of dysentery there is usually much wasting, and several conditions tend to produce this.

1. A loss, frequently repeated and extending over weeks, of albuminoid material from the bowel.

2. The constant aching and soreness and pain in the abdomen, so that the heart and breathing become weaker and weaker.

3. Hence great mental depression ensues, with loss of appetite.

4. The catarrhal inflammation spreads upwards from the colon to the stomach and œsophagus until it reaches the mouth. Therefore never fail to notice the tongue in cases of dysentery. The degree of catarrh in the mouth is an index of the intensity of the inflammation of the mucus membrane of the stomach. When the catarrhal inflammation has spread upwards in this way, the digestive power of the stomach and the absorptive power of the intestine are evidently greatly impaired, and therefore there is extreme wasting of tissues. In such cases the pulse is commonly very small, and the temperature is sub-normal, the vitality being very low. Now if the catarrh extends from the pharynx into the larynx, which is revealed usually by very harassing cough, and thence extends down the trachea, and bronchitis comes on, we can understand that very little bronchitis kills such exhausted patients.

Patients very commonly recover from dysentery, and whilst doing so the liquid fæcal evacuations may be passed for weeks. A patient may come to you complaining of chronic diarrhœa, and you should not overlook that this may be the result of dysentery and a stage in the recovery from that disease. That diarrhœa may continue for months, and ultimately the patient may entirely recover.

It is well that I should also tell you that I have known the catarrhal inflammation of the bronchi, which has resulted from protracted dysentery with great exhaustion, determine phthisical destruction of the lungs which killed the patient.

Here I will speak to you of another form of protracted diarrhœa—malarious or miasmatic diarrhœa, I do not quite know which to call it. For convenience some pathologists consider as malarious diseases those which arise in connection with decomposing vegetable matter; as miasmatic, those which arise in connection with decomposing animal matter.

This form of diarrhœa I have witnessed in sailors or others who have been admitted to the hospital immediately on returning from a voyage, and in a few of these cases the watery fæcal evacuations have continued from week to week until the patient died; and when I made the post-mortem examination I found no sign of dysentery and no morbid changes to account for the diarrhœa; I found merely an anæmic appearance, no gross organic disease of the bowel.

But of chronic diarrhœa not traceable to any gross disease or other recognisable morbid condition, there is much to be said. I remember a patient who had lived in China, and had made a fortune there. He had returned to this country some years before I saw him, and had, as people say, "everything a human creature could want"—excepting a comfortable belly—and he suffered from this form of uncontrollable diarrhœa until it killed him. He was seen by many medical men, but not one of them could define the cause. I mention this to indicate that probably many causal conditions combined to determine the result, not overlooking what is spoken of as worry.

LECTURE IX.

TYPHUS FEVER.

Typhus fever differs from typhoid fever in its course, its symptoms, and its morbid anatomy, and it is now also agreed that it has a different origin. Typhus fever in recent years has been comparatively rarely met with in this country. It was formerly known as famine fever, because, in years gone by, it was exceedingly common among the labouring classes who were huddled together in unhealthy dwellings, and were often much in want of proper food. It was exceedingly prevalent at the time of the potato-famine in Ireland. Typhus fever resembled relapsing fever in being found when there was great scarcity of food, where there was much overcrowding, and where there was much decomposition going on in organic matters; and when we see it now, we can usually ascertain that it has arisen in such like conditions.

In marked contrast with typhoid fever, typhus fever is usually ushered in abruptly by an attack of shivering; prostration comes on very rapidly; and within a few hours after the shivering, the patient is so weak that he is unable to get out of bed. With that prostration we see signs of venous congestion. The countenance becomes dusky, and almost livid. Soon tremor becomes a marked symptom; the tongue, the lips, and the limbs tremble, and we often see jerky movements. The patient becomes so prostrate that he lies on his back, and has difficulty in drawing up his limbs. The temperature is high, and with the dusky appearance of the face there is a dry, brown tongue; the lips also are parched, and covered with sordes. While the so-called rash of typhoid fever is not found till the second week of the disease, the rash of typhus fever appears in the first week—it may be as early as the second or third day. Another distinction from typhoid fever is the constancy with which the rash appears in typhus fever. We fail to find the rose-rash in about 25 per cent. of the cases of typhoid fever, but in typhus fever the so-called mulberry-rash is rarely absent—some physicians of large experience say that it is never absent.

Owing to the bluish-red mulberry colour of this rash we may mistake it for measles; but, unlike measles, it is little marked on the face, we find it chiefly on the trunk. I can recall an instance in which I was called in consultation to decide whether the patient was suffering from malignant measles or typhus fever. The rash was indistinguishable from that of typhus fever, but there was much bronchitis in the first few days of the attack, and from this I concluded that it was measles, and the progress of the case supported that view. Bronchitis in typhus fever comes on late, whilst in malignant measles there is bronchitis very early in the disease.

The rash has a mottled, mulberry-coloured, spotted appearance, and it is difficult to distinguish it at first from the mottling of the skin common in various feverish conditions. We sometimes have to ask, "Is this the mottling due to congestion of the skin common enough when the temperature is high, or is it the specific rash of typhus fever?" If you are in any doubt, wait a few days before giving a decided opinion. As the case advances the rash becomes much more pronounced; it is commonly thickly scattered all over the body, and least marked on the face.

There may be diarrhœa in typhus fever. Dr. Barlow, then senior physician to Guy's Hospital, used often to talk to me about that diarrhœa, and he was inclined to consider that diarrhœa was as common in typhus fever as in typhoid fever. But in these older observations it is to be remembered that years ago typhus and typhoid fevers were regarded as one disease, until Sir William Jenner called attention to the fact that they were two distinct fevers, and differentiated the symptoms of each. In both fevers diarrhœa is common, but in typhus fever, when there is diarrhœa, the stool has not the pea-soupy character of the typhoid stool; it is merely a watery fæcal evacuation.

In typhus fever the lungs become congested, so that in the first week the breathing is increased in frequency, and is labouring. The congestion of the lungs is also manifested by the excessive action of the diaphragm, and there is a dull percussion note over the posterior part of the lungs, with some crepitation, and we may estimate the severity of the attack by the signs of pulmonary congestion; it is the failure of breathing that is the immediate cause of death. As the lungs become more and more fatally affected the signs of pulmonary congestion become more and more marked. The crepitation increases, and the dulness on percussion is more and more extended upwards, the diaphragm has more difficulty in acting, and the pulse becomes softer and softer. There is a good deal of delirium of a low muttering kind, with increasing tendency to coma.

In all these feverish conditions we may find some amount of albumen in the urine, the amount depending on the extent of venous congestion in the kidney. We must not be discouraged by finding as much as even a half of albumen. I will not attempt to explain how it is that in some cases we find this large quantity of albumen, and in others none at all.

In the second week the severity of the symptoms usually much increases, and we ask ourselves, "Is the disease going to prove fatal?" Towards the end of the second week the prostration is most extreme; so great that it is difficult to get the patient to swallow anything. Paralysis of the bladder is common at this stage. Owing to the extreme prostration many die at this period of the disease.

In other cases we see a remarkable change which is common to typhus and relapsing fevers—both fevers which cause very profound prostration; what is known as a crisis rapidly occurs. On the fourteenth day, when the fever is at its height, and all the symptoms are most severe, and the case seems desperate, there is a sudden fall of temperature, perhaps to normal, in a few hours, a profuse sweat comes on, and the patient seems to be lifted out of a tremendous deadness, out of stupor and smokiness (the old word smokiness seems to be about the best); the lividity disappears, and the alteration in a few hours is simply astounding. In this short time the patient becomes able to turn in his bed and to answer questions clearly. Another remarkable feature of typhus fever, one in which it presents a strong contrast to typhoid fever, is that the patients pass through convalescence rapidly.

We cannot be certain from post-mortem examination alone that a patient has died from typhus fever; here again is a striking difference from typhoid fever. We find after death from typhus fever nothing more than this—the lungs are congested, they may be almost black, and weigh much heavier than normal, the spleen is congested, large, and black, and there is general venous congestion of the other organs. If we can obtain no clinical history we may have to consider, on making a post-mortem examination in such a case, whether the cause of death has been epilepsy. Even supposing that there is a history of a feverish attack we must remember that the patient may have died from an epileptic outburst during a feverish attack, the cause of which is obscure. In medical practice it may happen that we can obtain little or no history, the body having been found by the police, and on post-mortem examination we find the spleen exceedingly soft, the lungs congested, the body not extremely wasted; and there may be no history at all, or we may merely be told that the patient passed into a condition of stupor and rapidly died. The question arises whether it is a case of opium poisoning, of typhus fever, or of brain disease, or even of one more condition, it may be death from acute diabetes. That occurs exceedingly rapidly in some instances. It will assist you if I mention here that I remember a very impressive instance of that. A young woman was one day standing and talking to me easily and quietly, and I observed that she was fairly nourished and had a natural colour, and I knew that she was not thought to be dangerously ill in any way; but she had sugar in her urine, and was suffering from diabetes. On the evening of the same day she was seized with cerebral symptoms and died in a few hours. At the post-mortem examination we found the organs venously congested, nothing more.

But if we can obtain a history there is little or no difficulty in determining the cause of death. When it is due to typhus fever the condition of the spleen is usually a guide to us. It is exceedingly soft, and very black and large. We usually also find the walls of the heart very soft, and there is extreme congestion of the lungs. With such appearances, and such a history as I have described to you, there can be no doubt that the cause of death was typhus fever.

LECTURE X.

TYPHOID FEVER.

Typhoid fever may be so mild that we have difficulty in pronouncing it definitely to be typhoid fever; the doctors who see the case cannot agree as to its nature. On the other hand, typhoid fever may be so severe that the nature of the case is only apparent to a man of large experience. Even on post-mortem examination the same difficulty may arise. In a very severe and rapidly fatal case it may be difficult to recognise on the post-mortem table the characteristic changes of typhoid fever. The majority of cases lie between these extremes, and we have little or no difficulty in recognising their nature, but recognition only becomes possible as the case develops. Let us keep this clearly before our minds. We don't want to be hurried for the sake of cleverness, which destroys confidence and gets us into a difficulty. I repeat, typhoid fever can only be recognised as the case develops. Hence it is that some of the greatest mistakes in diagnosis are made in connection with typhoid fever. I will give you some illustrations of that.

The superintendent of one of the largest asylums in this country was called to see a gentleman, and he told the friends that the patient was insane; they must get him out and drive him about the country. Shortly after this he had pea-soupy motions, and in a fortnight he died from typhoid fever. I was called into the country to see that man: by the time I saw him the disease had developed, and there was not a shadow of a doubt that it was typhoid fever. At first sight it seemed that the doctor must have been careless. Not so. He was misled by knowing that there was insanity in the family, and he paid too much attention to the nervous disturbance.

A medical man called me in consultation one morning to see a patient who was shaking, with cold sweats, and evidently dying. This was on Monday. The Thursday before, he told me, the man had been at his work as a clerk in the city. On Friday he had some spots in his throat; these were touched with hydrochloric acid, under the idea that they were diphtheritic. The day after I saw him the man was dead. I brought the intestine to the hospital and showed it to the students; there was typical typhoid ulceration. I asked, "Didn't he say anything about being ill when he came home from his work at night, before he actually took to his bed?" "Oh, yes," it was answered, "he used to say, for days before he took to his bed, he was so wretched and weary, and that as he drove home at night he felt so weak he could hardly sit upright." Typhoid fever at the outset then may present such symptoms that it may be mistaken for diphtheria, scarlatina, or for other fevers. One of the first symptoms noticed in some cases is the appearance of a red rash on the skin, and the medical man wonders if the case is one of scarlatina, and it may be a day or more before he can satisfy himself that it is not.

One of the surgeons at Guy's had a man under his care for stricture; after being treated for this, he got up and walked about the ward like the other patients, and he was discharged as cured. He was walking away from the hospital when he was seized with a pain in the belly, and died rapidly from collapse. I saw Dr. Wilks make the post-mortem examination, and it was a case of typical typhoid ulceration of the bowel. I have seen cases treated by physicians in this hospital for pneumonia, which proved on post-mortem examination to be typhoid fever.

On the other hand, I have seen cases thought to be undoubted typhoid fever, turn out on the post-mortem table to be ulcerative endocarditis. Patients with supposed typhoid fever have been sent to a fever hospital, and they have been suffering from uræmia. Bearing these cases in mind, and others you will see for yourselves, you will know that you must not give a definite opinion until you are sure.

Typhoid fever occurs, like other fevers, in mild forms in which the symptoms are so little marked that the medical men cannot agree whether to call it typhoid fever or not. It is usual to speak of these cases as abortive typhoid fever. We are the more constrained to recognise the nature of such cases because in them sometimes perforation of the bowel suddenly comes on; the symptoms have been so mild that we do not learn till too late that we have to do with typhoid fever. In an intermediate group of cases, by far the largest, the symptoms are of moderate severity, and, given time, their origin is easily recognised. In the malignant form typhoid fever kills in the first or second week, before the symptoms can outwardly be much pronounced.

In all these cases, mild and severe alike, there is an obscure initial stage in which the symptoms are languor and weariness, headache and giddiness, increasing weakness. The symptom of symptoms is the utter weariness—some change rendering the patient incapable of keeping his mind and feeling occupied about his usual affairs: that is the most noticeable feature of this obscure state, which may last for a week or two.

In the mild cases the temperature rises a little, it may be 100° or 101° in the evening and lower in the morning. The appetite fails, and the inclination is to lie about. The tongue becomes somewhat furred, and this is more marked from day to day, and each day the temperature rises higher. This may go on for from eight to fourteen days. There may be a little looseness of the bowels, alternating perhaps with constipation. At the end of ten days or a fortnight the symptoms subside, and we say, probably it has been typhoid fever, but we have to admit that we are not sure. Then comes the question with regard to the taking of solid food, and how soon the patient may go to business. The answer must be given cautiously, for there may be more disease in the bowel than we should have thought from the slight nature of the attack. Once I opened the intestine of a patient who had died from typhoid fever and found three or four ulcers. I was struck by the fact that there were so few, but one of them had perforated the intestine and killed the patient. You see that we must go cautiously; get to the taking of solid food by degrees, and wait a week or so before the patient goes to work.

In more pronounced cases of typhoid fever the symptoms ushering in the disease may be of a more marked character.

For instance, there may be cerebral disturbance, delirium is a predominant symptom with a raised temperature; in other cases it is bronchitis with a raised temperature, and we wonder if it will turn out to be broncho-pneumonia. In other cases, intestinal symptoms are found; with the fever, diarrhœa comes on early in the attack, and severe diarrhœa. Never forget that. I will tell you of a sad experience I went through: a seaman just come home was not feeling well, and he went into the City and saw a medical man; he was given a strong aperient which purged him severely. I was called in to see the man. He was a strongly built sailor, lying in bed, quite calm and collected, no delirium, no pulmonary disturbance, but his belly was distended. The abdominal distension, the pea-soupy motions, and the high temperature, indicated much disease in his intestine, and I thought he would die. And that is why I told you to be sure not to overlook the fact that the intestinal symptoms may come on very early. At the time that his intestines were charged with morbid deposit he was given an aperient, and his intestine became so much affected that he could not survive. In other cases it is not the mucous membrane of the bowel but the mucous membrane of the throat that rivets our attention: patients come to the doctor complaining of sore throat, and, as I have already told you, there may be a red rash on the skin, so that it is easy to mistake such a case for scarlatina.

We notice, however, as the case advances that the typhoid symptoms become more pronounced. With the temperature keeping high day after day, the tongue becomes more parched, it gets brown and glazed down the middle, sordes begin to collect about the lips, there is increasing prostration, there is more and more delirium tewards night, and we say there is a typhoid condition—typhoid fever. The frequency of the breathing is increased, and, on listening over the bases of the lungs behind, we hear moist rales; on percussing over this area we find some degree of dulness, and we say that the bases of the lungs are congested, that there is an excess of blood in the lower parts of the lungs.

These symptoms become more and more marked towards the end of the first week, and up to this time usually there is little or no distension of the belly. If there is much distension we know that some morbid condition has lessened the contractile power of the intestine, allowing the gases to accumulate excessively, and we say that this morbid condition is the typhoid deposit in the intestinal wall. Usually the distension is not pronounced until the second week. If there is much diarrhœa the increasing discharge prevents the gases from accumulating; that is the great use of diarrhœa, it prevents a fatal tension in the intestine. When I was young I was much struck by hearing one of the senior physicians at Guy's Hospital say, "Typhoid fever cases with diarrhœa do the best." It was a revelation to me at that time, because I had been giving chalk, logwood, and other astringents. Here let me say that I cannot recall one case of typhoid fever in which diarrhœa was the cause of death. I have seen cases in which there has been extreme diarrhœa and wasting, but the patients have survived. But I have seen fatal cases with constipation; obstinate constipation is a much more dangerous symptom than recurring diarrhœa, even profuse diarrhœa. Where there is obstinate constipation it reveals that the bowel is much paralysed.

After I have put that experience before you, you will be prepared to understand why it is that the Germans lay so much stress on the treatment of typhoid fever with daily doses of calomel; there is a great deal of sound experience in this treatment, but the drug requires to be used with great caution. Do not let us overlook that in typhoid fever, as in dysentery and in flux from the bowel, there is a great tendency for the contents of the bowel to undergo rapid decomposition, and to become charged with fungous growths. In examining cholera stools, I used to find much fungous growth, and we find the same in diarrhœa and dysentery; now, the presence of bile tends to prevent such decomposition, bile being a powerful antiseptic. The old housemaid who used to clean the carpet with ox-bile knew what she was about. Therefore we look at the motions in cases of typhoid fever to see if they are yellow and contain bile. Now, there are two periods at which these stools contain bile: unquestionably they do so at the outset, when the diarrhœa is coming on, and they do so again when the case is subsiding—in the beginning of the first week, and towards the end of the third week. At the outset the discharge from the bowel is a liquid fæcal bilious evacuation; in the second week the liquid fæces become more ochrey-looking; towards the end of the second and about the beginning of the third week that pea-soupy character diminishes, and the stool has again a more bile-stained appearance. We know then that the condition of the intestine is improving, that the outpouring of serum is lessening, and that it is returning to its normal state. At any time there may be some blood in the evacuation; if there is not much we need not pay particular attention, but we do not overlook it, for it reveals to us that the morbid process is probably attended with a good deal of congestion; in other words, we learn that it is very acute.

In the second week we usually notice that the breathing is getting quickened, and that it is labouring; we notice also that the volume of the pulse is much diminished, and we feel it carefully. If its waves of energy are regularly repeated, we say that regularity means staying power; no matter how feeble, regularity means staying power. The pulse at this stage is naturally small and soft, but if it is regular we are encouraged to think it will go on. We hesitate to lift the patient up on account of the nervous agitation it produces, but we listen carefully at the front of the lungs, and the degree of harshness of the breathing there enables us to estimate the amount of congestion in the back of the lungs. We listen attentively for crepitation, to ascertain how much the bronchial tubes are closed up by muco-purulent matter. We pass the hand over the epigastric region, that we may feel for the pulsation of the right ventricle transmitted through the abdominal wall, for in this way we can estimate the degree of distension of the right side of the heart. I used to hear Dr. Wilks say, "Typhoid fever kills by pulmonary failure." We must never overlook the risk of pulmonary failure; we must listen to the breathing and watch the action of the diaphragm; the degree of action of the diaphragm usually reveals to us the amount of prostration.

This respiratory difficulty usually becomes greater and more dangerous as we pass from the second to the third week. Now we notice also that the patient is very tremulous, and is easily agitated. All his movements, including those of speech, are attended with much nervousness; they are tremulous and jerky. In cases of typhoid fever we have been accustomed to watch very carefully the amount of tremor, because it measures the degree of nervous prostration, and when the tremor is extreme patients commonly die from nervous prostration leading to fatal failure of the breathing. In this state there is active delirium. When the cases, formerly classed as continued fever, were separated into typhus and typhoid, English medical men used to lay great stress upon this, that extreme tremor, profound prostration, and low muttering delirium pointed rather to typhus than to typhoid fever. In some cases of typhoid fever, however, in which the prostration is very profound, the delirium is low and muttering; but the more characteristic delirium of typhoid fever is a much more manifest delirium—that which is termed active delirium; the patients become extremely restless, they struggle to get out of bed, are very self-asserting and inclined to chatter most of the night, and they may die from the exhaustion resulting from the activity of the delirium. This is, in fact, a frequent cause of death. It is difficult to get these patients to swallow their food, the heart becomes weaker and weaker, the breathing becomes more laboured, cold sweats come on, and they sink completely exhausted.

If I were going round the wards and showing you cases of typhoid fever, I should point out to you how they can be put into three groups: this is a matter of common experience. First, cases with the pulmonary symptoms much marked; secondly, those with the intestinal symptoms much marked; thirdly, those with the nervous symptoms, including delirium, much marked. Now, in making post-mortem examinations on cases from all these groups, I have been much struck by the fact that in all alike the intestine was much affected by typhoid deposit. This brought me to see that the localisation of the symptoms must depend on individual characteristics rather than on peculiarities in the disease. From this I have been led to learn something about the treatment of these conditions. I will take a case in point. I can almost see the man lying in the bed: a finefeatured, thin-lipped, fine-skinned young man, evidently of a very sensitive nature, tossing restlessly about. "If," I thought, "he is so restless at the beginning of the second week, what will he be when his brain is more sensitive and anæmic in the third week? What will happen when his intestine is on its extreme trial?" Well, about that time he was fighting for life. I believe that what saved him was this: that, recognising where his struggle would come, I had explained to the nurse that she must soothe him, so that he might get sleep. Often in typhoid fever a nurse helps so much in this, that she can soothe the mind when opium would deaden too much. I remember another case, that of a very sensitive man, and my house-physician said to me, "This is a mild case." I put my finger on the pulse, and was struck by the fact that it felt more like a piece of cord than an artery. I said to myself about him, "What will happen later, if he does not get rest?" He nearly died from perforation. Well now, what I want to impress on you is this, that whenever there is great nervous prostration the intestine has commonly much difficulty to deal with. Many people of sensitive nervous nature are liable to have attacks of diarrhœa brought on by fright, anxiety, or mental strain.

In the third week the typhoid deposit which has formed in the Peyer's patches and solitary glands, and it may even be in the large intestine, is as a rule clearing away. Then it is that sloughing commonly comes on in these parts, and thus the severity of the intestinal disturbance, with the pulmonary difficulty and the nervous exhaustion, kills many in the third week or in the beginning of the fourth. At this period also hæmorrhage is most liable to come on. If there is hæmorrhage and we are estimating the danger, we are guided by the suddenness of the onset, by the amount of blood lost, and by the repetition of the attacks. I say first by the suddenness. In the days when bleeding was commonly practised, if ten ounces of blood were taken away very suddenly, the patient fainted, while as much as forty ounces could be taken away gradually without loss of consciousness. Does then the blood flow out so suddenly that the patient, who, at the end of an attack of typhoid fever, has so little healthy blood flowing through his brain, is threatened with immediate death from shock? Many patients, however, lose much blood and recover, but in these cases there are one or two discharges of blood and then it ceases; that is why I told you we had to consider repetition in estimating the danger. In fatal cases there is usually repeated hæmorrhage. What we have then to consider in the treatment of these cases is how to prevent repetition. How are we to do that? I have been led to think that safety lies in keeping the blood flowing as freely as possible through the vessels of the intestine. If we let the blood stagnate about the slough and thus increase the tension, the patient will bleed to death. Owing to the typhoid deposit and to the sloughing conditions, the venules are very weak. Stimulate the breathing movements, and stimulate the action of the venous system; keep the window open, don't let the patient be covered with much clothing, let the air get to his skin; don't be afraid of his catching cold. You want to make him last for forty-eight hours, to prevent him from bleeding to death. I have seen drugs like tannic acid, gallic acid, and sulphuric acid given often, but my impression is that they produced no more effect than so much water would have done. You distribute five or ten grains of tannic acid all over the body; how is that going to stop up the little holes in the vessels through which the blood is running out? I could discover no rational treatment, but I have been encouraged to find that many patients who have hæmorrhage recover none the less, and I say "Don't let us be too anxious."

Never let us overlook this, that a patient with typhoid fever may bleed to death before there has been time for the passage of any blood per rectum. How are we to know that hæmorrhage is going on? If in the third or fourth week of typhoid, when we know that the typhoid deposit is coming away, and that there are naked ulcers in the intestine, there is a rapid fall of temperature, faintness, whiteness, with a cold sweat on the skin, we know there is rapid internal hæmorrhage, and that is the greatest risk of all. These are the most gloomy cases with which we have to deal. What does pathology teach us to do, then? It has taught me to recognise this, that if the depressing influences in the belly become extreme, there is a risk of death by failure of respiration. What shall we do in these cases?

Don't let the intestine get too much packed with gas, blood-clot, &c., for if this occurs, the breathing will get more difficult, and the risk of continuance or of recurrence of the hæmorrhage will be greater. I should not hesitate to order warm-water injections into the rectum immediately. I should try to keep down fatal tension in the belly; if it does nothing else, it will comfort the poor patient. I think that in these cases there would be no objection to the use of a little oil to keep the rectum working freely.

We should direct our minds a good deal to the smooth working of the sphincter ani; some of the most intractable cases of diarrhœa which have come under my notice have been due to an irritable condition of the lowermost part of the rectum. If there is much nervous strain in the lower end of the rectum it leads to venous congestion, and this to frequent desire to go to stool, and this tends to harass the upper part of the large intestine very much. Therefore, in typhoid fever, it will be well to look to that part, and, if it needs soothing, to give a warm-water or an oil injection. It is a useful practice in many cases of typhoid fever, both when there is diarrhœa and when there is constipation, to give an injection of a pint of warm water every morning, through a soft catheter with a funnel attached.

I can give you a useful guide: when you wish to measure the amount of venous obstruction, how far the breathing is failing, and to what extent the right side of the heart is getting over-distended, examine the inferior hæmorrhoidal veins and see if they are very full of blood. Piles and vesicular emphysema go together. Whenever there is difficulty of breathing the hæmorrhoidal veins get over-distended and piles tend to form. I have told you that in typhoid fever the great risk is from failure of the breathing.

You know that in the pelvis there are large venous plexuses; these in typhoid fever, when the breathing is failing to any extent, are apt to become overcharged with venous blood; thus the nutrition of this region suffers, and the power of the muscles is weakened, hence the bladder tends to get paralysed, and it is often necessary to draw off the water from time to time. You should never forget to ascertain the condition of the bladder, for you must remember that the bladder may become distended and the water may then dribble over, so that a patient whose bed is constantly wet may have thirty or forty ounces of urine in the bladder all the time.

Further, owing to the weakness of nutrition and to the pressure on the sacral region from lying on the back, there is a tendency to the formation of bed-sores. The time when these are most dangerous is usually in the stage of convalescence; therefore, we carefully examine the sacral region from time to time, and we take measures to prevent the formation of bed-sores, and to limit their extension when they have formed. In some cases they may extend so rapidly and so deeply that in a short time the ligaments on the posterior surface of the sacrum are exposed. I have known in such cases this happen; the typhoid symptoms had entirely disappeared, but the patient died in the fifth or sixth week from exhaustion from the bed-sore. At the post-mortem examination I found that the lungs had recovered and that the intestine was healthy, and that death was due to the exhaustion consequent on the bed-sore. In the fifth or sixth week the nervous system is in such an unstable condition that the irritation set up by the bed-sore is enough to cause death.

During the fourth and fifth weeks, if the attack has been severe, the condition of weakness is slow to disappear, the pulse remains very weak, the body is very much emaciated, and there is much restlessness at night; we need not, however, be discouraged by such symptoms, for patients who have got so far commonly recover.

There is another condition I must notice before I leave the acute stage, and that is hyperpyrexia. In the course of typhoid fever, but especially towards the middle or end, the temperature may rapidly rise to 105°, 107°, or even 110°. As this takes place lividity comes on, the pulse becomes exceedingly small, the breathing becomes labouring, and it is evident that the patient is tending to sink rapidly by exhaustion and failure of the circulation. Because the pulse is very small, because the breathing is labouring, and because there is lividity, we put the patient into a cold bath, or better, into a bath about, or a few degrees below, the body temperature, and cool it down to 70° or lower by putting ice into the water. As we do this we find that the pulse gets stronger and the breathing gets easier. As often as the threatening symptoms recur we repeat the bath, and in this way we may save cases which we should otherwise most certainly have lost.

Many cases of typhoid fever seem to be going on very well until the fourth or fifth week, and then from some obscure cause the temperature goes up again, the diarrhœa returns, the tongue gets dry and parched—in fact, there is a return of the typhoid fever process. Relapses are very common in typhoid fever. If you are asked, "What do you mean by a relapse?" say, "There is a return of the typhoid fever process, there are fresh deposits in the Peyer's patches and solitary glands, in fact, morbid changes similar to those of the primary attack." If asked, "How do you know that?" you should answer, "When we make a post-mortem examination we find naked ulcers from the first attack, and we find in other glands recent deposit very similar to that found in a primary attack in which death has occurred at an early period." The relapse usually lasts from eight to ten days, and the cases commonly recover. In rare cases there may be two, three, or even four relapses, until the typhoid fever process extends over months. During that time we have sometimes to deal with very curious nervous disturbances, twitchings, opisthotonos, harassing nervous cough, much mental aberration, &c., but with all that the patients may get quite well again. I remember being asked some years ago to see the sister of one of our students; she had been ill with typhoid fever for many weeks, having had relapse after relapse, and I was asked to see her because she had some of these peculiar nervous symptoms; but she got quite well in the end.

What conditions have we to apprehend after typhoid fever? There is sometimes much mental failure. We must always be very gentle with a patient recovering from typhoid fever, for the brain wastes as the body wastes, and a wasted brain becomes morbidly sensitive and exceedingly irritable and touchy; the mind may fail to such an extent that a patient may lie all day with fæces in the bed without complaining, and will rub his hand in fæces and carry them to the mouth. In other words, dementia comes on, but as the general nutrition improves and the circulation gets stronger he entirely recovers. After any acute disease with much wasting we may have to deal with a similar demented condition. Whenever there is great atrophy of the muscular system the brain commonly wastes, and as it wastes the mind fails. The patient may become acutely delirious, may lie and chatter from morning to night, may refuse food, may be extremely wakeful, in fact maniacal, and may die of exhaustion, but it is a condition from which he generally recovers.

There may be also a good deal of spinal failure, manifesting itself in weakness of the legs, loss of power over the bladder, and a great tendency to bed-sore.

Another common condition is protracted diarrhœa. The diarrhœa may return after an attack of typhoid fever for a year or two; if it does, do not look on it simply as an intestinal weakness, but try and discover the condition lying behind that weakness. I will give you two cases in point. A young woman had diarrhœa persisting for two years after typhoid fever. I did not then know her history, but I learned in after years that she became insane, and that there was recurring insanity for years, until she died; there was a history of insanity in the family. In another case a man had persistent diarrhœa after typhoid fever, and he was living a life which would exhaust any nervous system. We have to recognise the nature of these cases, for astringents fail, and we are puzzled to find out what is keeping up the diarrhœa; it is due to nervous conditions. After typhoid fever there may be failure also in the lungs.

I will sum up all this by saying, typhoid fever commonly leaves weakness in local or general nutrition, and that weakness may be manifested in any part of the body, it may be in the blood, as by anæmia, it may be in the lung, nervous system, or other parts.

What do we find on making a post mortem in death from typhoid fever? If the case is a malignant one, in which death has occurred in about the beginning of the second week, and the course has been characterised by rapid and extreme loss of strength, we find that the Peyer's patches and solitary glands are swelled and somewhat congested, and a practised eye (it requires a practised eye) notices in the swelled glands a grey, gelatinous, but not very translucent, deposit. We recognise at once the typhoid fever deposit, and putting together the clinical history and the appearance of the solitary glands and Peyer's patches, we pronounce definitely that the cause of death is typhoid fever. Fortunately those cases are not common.

More commonly death occurs in the third or fourth week. We always find the morbid changes most marked near the ileo-cæcal valve, in the lower part of the ileum; the other part of the intestine may be very little affected. We notice that the Peyer's patches and solitary glands are enlarged, and there is the characteristic typhoid deposit around the outer edge of these structures, but towards the centre the deposit has softened down and been discharged; commonly also we find that the deposit is yellow from bile-staining, and, moreover, that it is pulpy and evidently sloughing. Where there has been much delirium a very sloughy condition is common. Where hæmorrhage has been the cause of death, we find blood clots lying among the sloughs. If we examine the intestine from the peritoneal side, we find that the deposit has extended into the muscular coat, and even invaded the peritoneal membrane; this may have led to more or less peritonitis. We may find, in cases in which the sloughing has extended very deeply, that there is a little fæcal matter oozing through the wall of the bowel, that perforation has occurred. Then death may be due to widely diffused suppurative peritonitis, arising from the perforation.

We notice also that the mesenteric glands are congested and swelled; they are of a dark red colour, and contain a deposit similar to that in the wall of the intestine. The retro-peritoneal glands are in a similar condition, and to some extent also the bronchial glands; we have to recognise that there is more or less typhoid change going on throughout the lymphatic system, and this makes it easy to understand the great failure of general nutrition. I have once known suppuration occur in the mesenteric glands in connection with typhoid fever; the patient was a young woman, she had typhoid fever and seemed to be recovering, when peritonitis came on and proved fatal. I found suppuration around the mesenteric glands, and in these glands there was old calcareous deposit, and the irritation of the typhoid deposit had led to their suppuration.

We find the lungs congested and partly consolidated, and the patches of consolidation do not look like ordinary red hepatisation ; they are not granular, there is a homogeneous deposit with a peculiar mottled appearance ; this is the characteristic appearance of the pneumonia of typhoid fever. If the lungs are emphysematous there may be a good deal of muco-purulent exudation from the bronchial tubes, but the usual thing is to find these scattered masses of typhoid pneumonia. In some cases one lung may be consolidated almost throughout from apex to base ; these are the cases I have spoken of which clinically may be mistaken for ordinary pneumonia.

There is another organ in which curious changes occur in typhoid fever—the larynx. We may find sloughing typhoid deposit, like that in the intestine, about the aryteno-epiglottidean folds, and in some cases this may kill the patient by irritation of the larynx.

It is usual in books to speak of changes in the voluntary muscles ; what I have felt about these morbid changes is that they are common to protracted feverish conditions. The muscular fibre undergoes a granular disintegration, the transverse striæ are lost, the muscle cells have been converted into a granular substance, and with this there is much hyaline matter ; these are what are described as the typhoid fever changes in muscle.

To return to the description of the intestine, the morbid changes, although always most marked in the lower part of the ileum, may extend in a scattered manner right through the ileum, and even into the jejunum, but I do not remember ever seeing them as high as the duodenum. The typhoid deposit and ulceration may also extend in the other direction, downwards into the colon, even as far as the rectum. After death during the acute stage of typhoid fever, we usually find a swollen, dark, congested spleen ; this state of the spleen is not peculiar to typhoid fever, but it is found in many feverish conditions. It is in part due to the great irritation of the lymphoid tissues, which leads to a congestion of the spleen with much cellular increase.

We are sometimes in this difficulty about the ulceration of the intestine in typhoid fever; we find in the ileum a number of ulcers, occupying the position of the Peyer's patches and solitary glands, but they are what we speak of as naked ulcers; there is no deposit around their edges, and the edges of the ulcers are a little turned down, showing that some healing process has taken place; the question is, what has been the origin of that ulceration? The body is much emaciated, and we think it has been a case of typhoid fever, but there is no history, and we are not sure. What are we to put in the death certificate? Can we be certain that these ulcers are not the result of the patient having swallowed irritant poison some weeks before? In poisoning by acid, we may perhaps find no ulceration till we reach the ileum, but the ulcers are not in the position of the Peyer's patches or solitary glands; usually also there will be only one or two ulcers; further, the body is not much emaciated, and the mesenteric glands are not much nor widely affected, nor is there likely to be any bed-sore. Summing up the case before us, we say, "The emaciation, and (possibly) the bed-sore tell us that the case has been one of long illness; we find ulceration in the situation in which typhoid ulceration occurs; typhoid ulcers present this naked appearance when death has taken place in the fourth or fifth week; and there can be no doubt that this has been a case of typhoid fever."

I omitted to speak about the signs of perforation. Whenever dangerous irritation is arising from inflammation of the peritoneum, there is great restlessness, a most characteristic symptom, and the pulse becomes small and corded. It used to be a favourite question in examinations, "What is the pulse of peritonitis?" Answer, "It is small, hard and wiry." It is small because the irritation is tending to arrest the breathing, and the blood is being cut off from the left side of the heart; it is hard and wiry because the middle coat of the artery is in a condition of tonic contraction. We find also that the belly is getting either distended or retracted, and that the temperature is falling rapidly—it may rise for a time at the onset, but as collapse comes on it falls. We have another great guide in the alteration in the countenance. Healthy respiration and healthy expression go together; in peritonitis, the face seems to shrink, the eyes are sunken, and the expression anxious; this is what we term the abdominal expression. Do not forget the restlessness. My house-physician came to me once and said, "I want you to come and see that typhoid fever case, he is so very restless." I went to see him and found him twisting and turning as much as he could in his exhausted condition; his belly was much sunken. Post-mortem we found typhoid ulceration and perforation in more than one place.

You will be asked, "What do you think is the cause of typhoid fever?" I will tell you what people think and teach about it. There is a growing feeling that typhoid fever is an infectious disease, carried from one person to another. And yet we take typhoid fever cases into the wards of a general hospital like this, as if there were no risk of infection. And speaking broadly I think it would be true to say that there is little risk of infection, but we have to ask this question, "May any poisonous material be carried from a typhoid fever patient to another person who happens at that time to be much out of health?" I am inclined to think that in typhoid fever there is a period of weakness many weeks before the febrile outburst, a period during which perhaps persons are liable to infection.

I will give you a valuable piece of experience. I have made many post-mortem examinations on persons who had died from cholera and from typhoid fever, and I have not found a single case in which there was Bright's disease or any organ much damaged. I was brought to this conclusion, that people who have serious organic disease do not get cholera and do not get typhoid fever. Many of these people, however, were bloated and fat, as if they had drunk hard, others had a little local damage, hence I came to this further conclusion, that the people who are weak and have a certain amount of blood in their bodies which can undergo rapid morbid change, are those who are subject to infection. A man has drunk hard and he gets pneumonia, and dies rapidly; there is imperfect co-ordination in his body, and he has a lot of dying blood ready to undergo fatal decomposition.

Typhoid fever is most prevalent in the autumn months, September, October, and November, this is established beyond question. There must be some reason for this seasonal prevalence. During the autumn rains, there is much and active decomposition in the summer's produce, dead leaves, and the like. At the same time there is much fæcal matter lying about. It has been supposed that the heavy rains wash the decomposing matters into the ground, and that the water is thus poisoned. We have here carefully to distinguish between the facts and the theory which has been put forward to connect them, for we have other facts to consider. In one case there was a quantity of fæcal matter buried up, and it came from a source where there had been typhoid fever. It was opened up, and many of the men engaged in the work sickened with typhoid fever. This is very instructive. We used to find that the people who got cholera were the women who washed the patients' clothes, not the nurses nor the medical men. The poison had been lying and had decomposed. Typhoid fever and emanations from cesspools and soil-pipes have been found to be connected as cause and effect. I once had this remarkable experience. I had to visit a house in Shoreditch, from which five persons had been sent to the hospital for typhoid fever. I found a nice clean house, I was struck by the cleanness; I found a well-paved yard, and I thought that this did not look like typhoid fever coming from dirt. The mistress of the house said to me, "When the house has been shut up all night, there is such a smell which seems to come from under the front-door." When that yard was opened up, there was a huge cesspool under those paving-stones; the poisonous gases were sucked up by the heat of the house. You might gather many similar facts, showing a connection between typhoid fever and covered cesspools in close proximity to a house. We do not find the same connection with cesspools in gardens. There is no more powerful disinfectant than the air, but then to allow of that disinfection, the air must be able to get into the ground.

Now let us look at the other side of the question. There are many places with a quantity of sewage matter lying about, but they are free from typhoid fever. I have been astonished again and again by finding black sewage matter under the floor of sleeping rooms, and yet no typhoid fever. "How is it that, while there has been a neglected cesspool there for years, yet there has been no typhoid fever until lately?" I do not fail to take into account that a man may have been there and left germs. I do not dispute this or his leavings, but I think we strain a little too much to prop up a theory.

We must recognise both sides. It is undisputed that when an army saturates the ground with ordure, typhoid fever and dysentery follow. This has been noted often in the Crimea and elsewhere. But how would it have been if the soldiers had not been harassed so much, had been properly fed, and in other ways so treated as to keep up their strength? You put the poor creatures into such conditions that their health has been failing for months, and when they die you blame the drainage! I have been brought very much to this conclusion, that there is a previous failure of health. The body of the President of one of our leading scientific societies is lying now in Westminster Abbey. I remember standing by his side some years ago and wondering what made him so careworn. I afterwards learned he died from typhoid fever; they thought he had taken it on the Continent, but his health had been failing for years before.

LECTURE XI.

DIPHTHERIA.

DIPHTHERIA is a disease in which there is severe general disturbance, accompanying a local inflammation of part of one or more of the mucous membranes of the body, the characteristic of this inflammation being the formation of what is known as a false membrane on the inflamed surface. The commonest seat of the local inflammation is the pharynx and fauces, and the disease is then termed pharyngeal diphtheria. Less commonly, the primary seat is the larynx—laryngeal diphtheria. In rare cases it may begin in the mouth or nose—buccal or nasal diphtheria; it may begin in the eye or on the vulva, and in very rare cases on the skin—cutaneous diphtheria. These cases of cutaneous diphtheria only occur in severe epidemics, and they arise from inoculation of some crack, abrasion, or slight wound with the diphtheritic poison. It has arisen on the cracked nipples of a woman suckling a diphtheritic infant; it has occurred on a blistered surface on the hand of the nurse of a diphtheritic patient. When diphtheritic membrane has formed on any portion of the skin it spreads rapidly. But I do not purpose to consider these rarities, and in this lecture shall speak of the commoner forms of diphtheria.

Directly we look at the throat of a patient with diphtheria, we are impressed by its extreme arterio-capillary congestion. It is not of a bluish-red colour, but rather a fiery red; the only other condition which produces a similar appearance of the throat is extreme local irritation, as from a scald. On the congested and swelled surface we soon notice grey patches of false membrane, and, as we watch them, from time to time we notice that they are spreading rapidly. The place where the membrane is first formed is most commonly one or both tonsils, or on the uvula or edge of the soft palate. The patches spread rapidly at their edges, and fuse, so that a complete cast of the fauces may sometimes be peeled off; if this is done, the membrane is re-formed in a few hours. On stripping off the membrane we see a granular and ulcerated surface, and this surface commonly bleeds freely, so that it is evident that the exudation is in the substance of the mucous membrane as well as on its surface. Do not let us overlook this fact, that the diphtheritic inflammation extends into the sub-mucous areolar tissue, and that its most active process is below the surface; for it teaches us that no superficial application can arrest it. At this stage the all-important question is, whether the inflammation will go into the larynx. I think you will understand better what I have to say about that, if I first describe to you the morbid appearances in a case of diphtheria which has proved fatal from laryngeal obstruction.

We find the mucous membrane of the fauces, pharynx, larynx, and trachea of a deep red colour, and containing numerous ecchymoses. It is covered with dirty grey patches of membrane; the membrane is whiter during life than we find it on the post-mortem table. It is commonly found on the tonsils, the soft palate, the epiglottis, the aryteno-epiglottidean folds; it entirely covers the vocal cords; it extends down the larynx and trachea in one continuous sheet, and even into the larger bronchi. But as we leave the larger bronchi we no longer find membrane. Where there is consistent membrane which adheres where it is poured out, there is usually, except immediately over the vocal cords, enough space left for the air to pass. But in the medium-sized and smaller bronchi we find a translucent glutinous substance which fills the tubes. Remember this fact in considering the question of tracheotomy. Underneath the false membrane we find a granular and ulcerated surface, in which the sub-mucous tissues are exposed.

We find the lungs, when we cut into them, of a pale colour, and rather grey, but they rapidly turn red on exposure; from this we learn that the air could not get to the blood. We also notice that the lungs are drier and weigh less than usual—

that is to say, there is less blood in them than usual. The blood could not get through the lungs, and accordingly we find the left ventricle contracted and empty, while the right side of the heart is over-distended; the venæ cavæ are very full, and there is extreme venous congestion of the liver, kidneys, and other organs. We see this always—that when the air cannot get to the blood, then the blood cannot get through the lungs, the left side of the heart and the arteries become too empty, and the right side of the heart and the veins become too full. We see this in death by drowning or strangulation, just as we do in death from laryngeal and bronchial obstruction in diphtheria. Note that I said we find venous congestion of the kidney, not nephritis. I have never seen the latter in death during the acute stage of diphtheria; if the patient gets better, and the venous congestion of the kidney passes away, then acute nephritis may come on.

I am not going to say much about micrococci and bacilli. I don't think that knowledge about them will help us much to treatment. When diphtheria was first recognised in England as an epidemic disease, it was found that the membrane contained fungous growths, spores, and mycelium. A commission was appointed by the Royal College of Physicians to inquire into the matter, and its report was to the effect that these fungous growths were the consequence of the disease, not the cause. Now we hear a great deal of these micrococci and bacilli. I think it probable that they, too, grow in the diseased structures secondarily; I think that the time will come when it will be generally recognised that these organisms are accessory and accidental concomitants, not causes of disease.

To return to the clinical signs. Sometimes the disease begins as if with a severe shock to the system, but this is unusual. As a rule the disease begins insidiously. It is sometimes remarkable how little soreness there is in the throat; for this reason, it is well to examine the throat in all feverish conditions, whether the patient complains of sore throat or not.

If the diphtheritic membrane is first formed in the larynx, extension to the pharynx usuallyoccurs; and conversely, if the membrane is first formed in the pharynx, extension to the larynx is common, but by no means an essential feature of the disease. It is obvious that the mere presence of false membrane in the pharynx is, from a mechanical point of view, of very little importance; whilst a diphtheritic inflammation of the larynx is fraught with the gravest consequences, and the matter is still more serious when the inflammation extends down into the medium-sized bronchi, in which a tenacious and semi-fluid exudation is formed, for tracheotomy is then unavailing.

What are the signs of the onset of laryngeal obstruction, and of the plugging of the bronchi with exudation? Watch the pulse. At the outset of suffocation it has been found that the arterial tension is raised; then there is an irritable knocking action of the left ventricle, with a full, hard pulse. As the right ventricle becomes over-distended, because the blood is unable to get through the lungs, the arterial tension falls. The arteries, containing little blood, and that imperfectly aërated, contract, owing to irritation, and become small; this makes the vessel feel like a piece of cord, and the pulse very small. We hear stridor in the breathing, due to the obstruction in the larynx. Extreme restlessness comes on, and there is increasing weakness. The face becomes ashy in colour; the urine is scanty, of high specific gravity, there is a great excess of urea, and it is charged with albumen. The amount of albumen is an index of the degree of venous congestion of the kidney, and this is a measure of the obstruction to the breathing. Our guides in estimating the degree of laryngeal obstruction are, then, the pulse, the condition of the urine, the colour of the face, and the laryngeal stridor. We learn much more from these than from the laryngoscope.

Then comes the question, "Shall I open the windpipe?" We might take up a fixed position here, but it is better to ask the question in another way: "When shall I not open the windpipe?" Say, "It's of no use to open the windpipe when the exudation is choking up the smaller bronchi. If, therefore, we find much crepitation through both lungs, a flat percussion note, signs of great distension of the right side of the heart, and an exceedingly small pulse, we know that the exudation is extending down the tubes, and tracheotomy is useless."

As the growth extends down the bronchial tubes the veins of the neck stand out; the epigastrium sinks in more and more during inspiration; the pulse becomes smaller and smaller, until at length we can no longer feel it. Respiratory movements may go on, not only for minutes, but for hours after the patient is pulseless, but the temperature of the body falls, the labouring breathing becomes weaker and weaker, until the patient dies of suffocation.

Recovery from laryngeal diphtheria, with or without tracheotomy, is not very common, but a few cases do recover; recovery is much more common in cases in which the false membrane is limited to the pharyngeal region. Before speaking of recovery, I have a few more particulars of the spread of the disease to give you. While the changes I have described are taking place in the throat the lymphatic system of the throat becomes much poisoned and congested, and we can often feel the enlarged glands behind the angle of the jaw. We can estimate the severity of diphtheria, as of scarlatina and other specific fevers, by the extent to which the glandular system is implicated. The inflammation may extend in other directions besides into the larynx. It may pass forwards through the nose, until membrane is seen projecting from the nostrils; but before we see this membrane, we know that the nose is affected by the glairy muco-purulent discharge from the nostrils, the smell of which is characteristically offensive. It may extend along the lachrymal canal to the eye, and thus give rise to one of the most serious forms of conjunctivitis. It may extend along the Eustachian tube to the ear, and lead to chronic suppurative disease of the middle ear, and even to absolute deafness, so that diphtheria in early childhood is one of the causes of deaf-mutism.

How are we to promote recovery? Do we learn anything in the post-mortem room which can guide us in this matter? Where there has been no extension into the larynx, or where such extension has not been fatal, the patient may seem to be mending for a day or two, and then broncho-pneumonia may come on and prove fatal; or death may arise from some secondary inflammation; or there may be rapid cardiac failure; or extreme asthenia may prove fatal. Now, in these cases we no longer find false membrane; the membrane has been softened down into a semi-fluid puriform material, and the ulcerated mucous membrane is bathed in pus. When we see that, we say to ourselves, "The natural tendency to recovery is by the formation of pus. These leucocytes burrow underground like rats, and eat up the poison; and yet for years it has been our practice to plaster the surface of the inflamed mucous membrane with caustics and astringents, so as to make it sticky and hard, and to stop the circulation."

Medical men have now come to see that this use of caustics and astringents is mischievous, because they check the natural efforts at elimination of the poison. The most active processes are going on in the sub-mucous tissues, and all our efforts should be directed towards promoting suppuration; we have to keep up the circulation, so that the leucocytes and serum may wash away the exudation. For these reasons we let the patient inhale steam, and we give him as much air as possible.

People say I am too fond of giving whisky, but I give it because I have found it useful. We want to keep up the circulation and soothe the brain, to prevent the brain from feeling the irritation too much. Why not use opium for this purpose? Because opium stays too long in the brain. You give opium in diphtheria, and an hour later your patient may be fighting for life with obstruction to the breathing, and the nerve tissues which carry on respiration are deadened by opium. Alcohol is eliminated more quickly, and does not deaden the respiratory centre in the way in which opium does.

After diphtheria there may be much anæmia. Whenever there is great difficulty of breathing, anæmia is liable to come on, but we see this anæmia also after cases of purely pharyngeal diphtheria. With that anæmia there is more or less fatty degeneration of the heart, and sometimes very sudden death from syncope. We have to remember that after diphtheria much of the blood may be dead. We must give much air and freshen the blood with fresh food. Do not be afraid of a little calomel now and again, nor of an open window. Our view should be to "make man fresh."

I have told you that in death during the acute stage of diphtheria the only change I have found in the kidney is venous congestion; there is exudation of leucocytes, but no inflammation visible to the naked eye. Two or three weeks after the acute stage nephritis may come—acute Bright's disease—and in these cases the prognosis is favourable, recovery usually occurs.

After diphtheria, it may be after a severe case, it may be after one so mild that its nature was doubted, the patient has made a good recovery. Then two or three weeks later some weakness steals over him, and we may suspect the beginning of phthisis. Then we are told that as the patient was swallowing, some of the fluid came back through the nose; then we know with what we have to deal—that the soft palate is paralysed. We touch it with something, and it hardly moves, for there is anæsthesia as well as paralysis. Next perhaps a slight squint is noticed; some of the ocular muscles are affected. There is a huskiness in the voice, due to partial paralysis of the laryngeal muscles. Then the patient complains of tingling sensations in the limbs, and we know this is a sensory affection preceding paralysis. Gradually the legs and arms become paralysed, it may be completely, so that the patient is unable to move hand or foot; it may be to a lesser degree, so that there is an uncertain staggering gait, while the finer movements of the hands are interfered with. Are the automatic movements, such as those of breathing, impaired? Very rarely, but I saw one case in a child in which at first the thoracic movements of respiration were checked, so that the diaphragm acted excessively, and the next day the diaphragm failed too, and the child died.

What are we to do in these cases of diphtheritic paralysis? In order, if possible, to prevent its onset, we should, after diphtheria, endeavour as soon as possible to restore healthy conditions of nutrition. We should let the patient be as much out of doors as possible, and keep the circulation active, so as to enable the healthy parts of the body to keep the morbid under control. When there is extensive paralysis we must keep the patient at rest; the prognosis is favourable; death is exceedingly rare, and the cases hardly ever become chronic, but progress is often slow, and much patience is required.

LECTURE XII.

RHEUMATIC FEVER AND ITS COMPLICATIONS.

RHEUMATIC fever may kill and on the post-mortem table we may find nothing—no morbid change to account for death. We merely find evidence that there has been some arthritis, but so little that we cannot attribute the death to it. Rheumatic fever is characterised by inflammation of the joints, but we find inflammation of the joints in other diseases, and it is not always an easy matter at first to pronounce whether a given case of arthritis is due to rheumatic fever, or to pyæmia, gonorrhœal rheumatism, gout, or rheumatic gout. We cannot then distinguish rheumatic fever clinically by considering the joint affection alone, and we have to ask what other conditions are characteristic of the rheumatic fever process? You will see that there are many when I tell you the way in which rheumatic fever may begin. In some cases the joints may be attacked at the outset, in others not till towards the end of the illness. I have known rheumatic fever ushered in with paralysis of the facial nerve, and whilst I was taking notes of the case I noticed that the skin had a sour smell; a few days later there was undoubted rheumatic inflammation of the joints. The disease may begin with paralysis of the sixth cranial nerve; it may begin with an attack of sciatica. Not uncommonly the disease begins with pleurisy; or there may be pleuro-pneumonia or pericarditis before there is any arthritis. One Friday, I remember, a young man came to me at Victoria Park Hospital suffering from sciatica; on Sunday he came to me at Guy's Hospital, and then I heard a pericardial rub. Two or three days later there was arthritis.

Before the joints become affected, and before there is any sign of local inflammation, rheumatic fever is commonly ushered in by anæmia, by dyspeptic symptoms, by increasing weakness, sweating at night, with the characteristic sour smell of the perspiration, and a slight rise of temperature towards evening. Now in this condition, although there is no arthritis, there is great risk of endocarditis. Some writers have contended that there is greater risk of endocarditis in such a condition than when there is well-marked arthritis; we certainly see many cases of severe heart disease in which there is no clear history of rheumatic fever, and yet on post-mortem examination we find unmistakable rheumatic changes in the valves, and we are led to think that the endocarditis must have come on sub-acutely in that anæmic condition I have described, and that the rheumatic attack subsided without having ever given rise to arthritis.

After this anæmic febrile condition has lasted for a time, it may be for a few weeks, the patient begins to suffer from stiffness in the joints, and the joints which are most worked suffer the most, usually those of the ankles, knees, wrists and elbows. After the stiffness comes pain, and then redness and swelling. But the general experience in rheumatic fever is that there is little effusion into the joints; there is a little fluid in the synovial cavity, but the swelling is chiefly due to inflammatory œdema of the cellular tissue around the joint. Rheumatic arthritis never leads to suppuration. You will tell me that your books say that there are occasional cases of suppuration; this is one of those matters in which the writers of books have the habit of copying one another without regard to clinical and pathological experience. In cases of multiple arthritis going on to suppuration I have usually found pyæmia, and where there was no pyæmia there were rheumatic gouty changes in the joints. Another reason why writers have not been clear upon this subject is that we so rarely have an opportunity of making a post-mortem on a primary case of rheumatic fever. In my whole experience I have only made two or three. Bearing in mind the simple nature of the inflammatory changes we can easily understand that the serum may be removed, and the joint left undamaged. One of the most remarkable features of the arthritis of rheumatic fever is that it is erratic. The inflammation may last for forty-eight hours in one joint and then disappear and attack another, leaving the first quite free from pain and stiffness. Where any permanent thickening has been left after an attack, it has been due to rheumatic gouty changes in the joint.

While the joint affection is going on like this we very commonly find that the chest organs are attacked by inflammation; the inflammation may begin on the pleura, extend to the lungs and then to the heart. It very commonly begins in the left pleura. It may begin in the heart, and extend by way of the pericardium to the pleura and lung. A common thing is for the patient to have at the same time pleuro-pneumonia at the base of the left lung and pericarditis, and sometimes also endocarditis. The heart may, as I said above, be affected by rheumatic inflammation before there is any arthritis, but most commonly the inflammation of the heart and the arthritis begin simultaneously.

We have now to consider a question of great importance; if the heart has not been affected during the first week of the arthritis, will it subsequently be attacked with rheumatic inflammation? I think that in such a case the heart will escape entirely, unless the patient has a relapse after the arthritis has subsided. I was led to this conclusion in this way. Some years ago I was studying the literature of rheumatic fever, and I examined the reports of many observers. Each of these writers had some favourite method of treatment: one advocated alkalies, another blisters, another lemon-juice. Now in each report there was a statement to this effect: "If the patient is free from heart disease at the time the treatment is begun, and if no heart disease develops during the first two or three days of treatment, if, that is to say, there is time to get him under the influence of the treatment, then the development of heart mischief will be entirely avoided." I thought that this was very curious, that such dissimilar methods of treatment should have the same result, and I wondered what it meant. Then I came to recognise this simple fact, that those observers were watching the natural course of the disease and attributing the results to treatment. I understood that if a patient does not get heart affection during the first week of the arthritis, it is most probable that he will entirely escape.

The onset of rheumatic pericarditis may be so obscure that the patient may walk about after the inflammation has begun. Usually we first detect it by objective signs. We are examining the heart and hear a pericardial rub; we are usually led to examine the heart because the patient complains of weakness and shortness of breath; if there is complaint of pain it is most commonly in the epigastrium. The gradual onset of symptoms seems to depend on this, that the inflammation does not come on very rapidly, and that it first attacks a very small portion of the pericardium, and spreads gradually. Sometimes, however, we have a very different experience with rheumatic pericarditis; it comes on with agonising pain very similar to that of angina pectoris—a violent griping, contracting pain across the front of the chest, binding breathing down to such an extent that the poor patient says, "Unless you give me relief it will rapidly kill me." On listening over the heart region we can hear nothing abnormal, but we think it is the onset of an attack of rheumatic pericarditis, for we know the order of the symptoms; moreover, there are probably signs of rheumatic fever, the patient is sweating, has a high temperature, and pain and swelling in the joints. The pain comes on in paroxysms like angina pectoris, and we cannot but call it rheumatic angina. In about forty-eight hours we hear a pericardial rub at one spot, and in a few hours more it is audible all over the front of the heart; as the objective signs become marked, the pain disappears. Now the inference we draw from that is this, that whilst the blood is accumulating in the vessels of the pericardium, and is distending them much, the tension gives rise to severe pain, but as the exudation is poured out the tension in the vessels is relieved, and therefore there is no longer any pain. Similarly, we find that in the joints in rheumatic fever there is most pain when there is least effusion. I remember hearing a tall, strongly built young woman say, "This pain in my joints will kill me unless you give me relief," and in her case there was very little swelling indeed. It did kill her in a very few days, and it was surprising on post-mortem examination to find how little effusion there was. Never let us overlook that in rheumatic fever the greatest danger of all comes from the severity of the pain, that the pain alone may kill; where the pain is not severe it is astonishing to see how many joints may be affected and swell, how much inflammation there may be in and about the heart and how much inflammation there may be in the lungs, and yet the patient recover. The greatest risk is therefore from the severity of the pain. With the pain are associated extensive changes in the brain and mind, and we have to consider how disturbance in the brain and heart disease come on together, and more or less determine each other. We have come to recognise that a silent delirium is much more common in rheumatic fever than we had previously thought. It is an unquestionable fact that easy working of the cerebro-spinal system and easy working of the cardiac plexus are associated; to prove this it is only necessary to watch carefully the action of the heart of a person when his mind is agitated. Then, again, it has been demonstrated that pain kills through fear, fear leading to paralysis of the right side of the heart.

In listening to the heart in rheumatic fever, before there was any murmur or any sign of pericarditis, I have been impressed by the tension of the beat, by the restrained nature of the sounds, indicating that the heart had lost its easy flowing beat, and tended more and more to give an abruptly constricted knock. Those who recognise endocarditis and pericarditis before a bruit or rub is audible, do so by noticing the irritability of the heart's action. There is an excitable irritable knocking of the heart against the chest wall, which, I repeat, is not an easy outflowing of energy, but a restricted movement. We may compare the healthy action of the heart to the easy onflow of a stream in an open channel, and the irritable action I have described to the uneasy disturbance which takes place in the stream when a log of wood becomes fixed in mid-channel. As this restrained action comes on during the severe pain there is a tendency to the excessive accumulation of blood in the right side of the heart, and we get signs of its distension; the breathing becomes quickened and disturbed, the venous system becomes overfull, and the coronary veins being distended, the blood is not able to flow freely through the wall of the heart, and the muscular substance, being ill-nourished, becomes enfeebled. We must keep watch for the early signs of heart failure; pallor of the face is a sign that the arteries are not properly filled, and lividity a sign that the venous system is becoming overcharged. We feel if there is any undue tension in the pulse, for, as I told you in my lecture on diphtheria, when pulmonary trouble is coming on, the tension in the arteries is raised. In this early stage we notice that the valvular actions are very abrupt and irritable, especially the pulmonary semilunar valve, telling us that the pulmonary artery is over-distended. You will ask me why I tell you all these things. Because we have to try and ward off endocarditis and pericarditis, and I want to teach you to recognise when they are threatening to come on; I have so many times seen the utter failure of merely recognising a mitral bruit and doing nothing.

As the wall of the heart gets overcharged with blood the tissues must tend to swell, and when we take this into consideration and reflect upon the disturbance in the vaso-motor mechanism, and other parts of the nervous system, we can get some idea as to how the inflammation comes on. The earliest sign of endocarditis I have seen has been a cloudy swelling of the substance of the valve; the valve loses its silvery translucent appearance, it becomes milky, and I have found under the microscope that it is charged with a fine granular substance. The fibrous tissue of the valve is so swelled by that granular material, that under the microscope we seem to be looking at a crumbling mass. As the inflammation develops we find accumulation of leucocytes, and in later stages we find successively, larger nucleated spherical cells, then spindle-cells, then a fibro-cellular substance; these are the successive changes of endocarditis. If the inflammation is extremely acute, red corpuscles are mixed with the leucocytes, and the valve becomes so much distended with the inflammatory products that it sloughs, and we get what is termed ulcerative endocarditis; in some of the most terrible forms of rheumatic fever we get this destruction of the valves by ulcerative endocarditis.

If the pain is very severe, and if in consequence the vaso-motor mechanism is much weakened, and there is venous congestion of the wall of the heart, we may infer that there is great risk of endocarditis coming on, and our first effort must be to prevent this by relieving the pain.

Rheumatic endocarditis usually begins on the auricular surface of the mitral valve, but in some cases it begins in the endocardium of the left auricle, perhaps an inch above the valve, and spreads downwards. We find the surface of the endocardium covered by the granular lymph I have described to you, and the mitral valve much swelled. The fibrous structure of the aortic flap of the mitral valve is continuous with that of the nearest flap of the aortic valve, and, further, the roots of the aortic valve are part of a kind of fibrous tendon round the base of the heart, so that it is easy to understand how the inflammation beginning on the endocardium of the left auricle may extend by way of the mitral valve and the aortic valve to the pericardium, and hence endocarditis is very commonly followed by pericarditis; it has indeed been maintained that some degree of pericarditis invariably follows endocarditis. Conversely the inflammation beginning in the pericardium may extend to the endocardium. The fibrous base of the mitral valve is also connected with the tricuspid valve, and the inflammation sometimes extends through the septum and leads to endocarditis of the tricuspid valve.

Endocarditis in rheumatic fever is usually revealed by a sound which tells us that the tension of the valve is altered; we usually first notice that the intensity of the first sound is much increased, and it is generally difficult for a time to decide whether we are to call the sound we hear a bruit or not, and we agree to call it a prolonged first sound, murmurish, and if later it becomes much more blowing we then call it a bruit. We must be quite ready to agree that there are many cases in which it is impossible to speak confidently about the existence or non-existence of a bruit. In many of these doubtful cases the bruit is soon unmistakable, but in many others the heart sounds become again quite normal, and as far as we can judge there is no permanent affection of the heart; for these reasons do not be too eager to give a definite opinion, but wait until you are quite sure.

As the endocarditis increases we notice that there is more shortness of breath, and as we know that endocarditis kills by failure of the breathing, by the lungs becoming fatally congested, we measure the severity of the endocarditis by the degree of dyspnœa. We do not measure it simply by the intensity of the bruit, though this undoubtedly tells us the alteration in the tension of the valve, for we have learned that in many cases of endocarditis with a loud bruit there is little or no dyspnœa as long as the patient is lying quietly on his back; we may, indeed, notice that the breathing is somewhat quickened, but the patient does not complain much of difficulty of breathing. Now the alteration of the tension of the valve, and hence the intensity of the bruit, certainly do depend on the extent of the endocarditis, so you see I cannot have been speaking accurately when I said that "endocarditis kills by failure of the breathing." If with a very loud bruit there may sometimes be little dyspnœa, and conversely with a scarcely audible bruit there may sometimes be very much, and even fatal dyspnœa, it is obvious that there must be some other condition than endocarditis, strictly speaking, to lead to fatal failure of the breathing. Now what is that condition? This is a question which has much occupied my mind. I have been guided by what I have found in the post-mortem room. I have opened a heart and found a small area of endocarditis, and have been told that the patient's breathing had failed rapidly. On the other hand, I have made a post-mortem examination and found gross changes in the valves, obviously the result of extensive and severe endocarditis, and have been told that it is many years since the patient's heart first became affected. How is it that in the one case the small inflammation has killed rapidly, whilst in the other, with all that inflammation of the endocardium, the heart has been able to keep up the circulation for years? On examining the heart's wall more carefully in the cases of endocarditis in which death has occurred rapidly I found that the inflammation had spread from the mitral valve into the substance of the heart—that is, that there had been myocarditis as well as endocarditis. Then it became clear to me that when this inflammation was going on in the connective substance of the heart it was impossible that the nutritive juices needed by the muscular substance of the heart could be properly formed, and the muscular fibre had failed. I learned that the danger in these cases, the fatal failure of the breathing, depended not on endocarditis and changes in the valves, but on the degree to which the heart muscle was affected by inflammation. Before continuing this subject it is necessary to return to the consideration of that of pericarditis.

You will remember what I told you about the extension of inflammation through the ring of fibrous tissue at the base of the heart; in accordance with this we find that rheumatic pericarditis begins usually at the base. We often find very great difficulty in determining whether a patient is suffering from endocarditis or pericarditis; we hear a blowing sound at the base of the heart, the character of which makes us think of endocarditis, but the position of pericarditis. The early sign of pericarditis may then be a soft blowing sound, but soon there is mixed with it a creaking sound, which has been called the new leather creak of pericarditis. As lymph accumulates the creak becomes a rub, a friction sound, and soon occurs both with the systole and the diastole of the heart, with a slight and sometimes incomplete pause, and we get what is known as a to-and-fro friction-sound; by this time the lymph has extended to the apex and we hear this friction-sound all over the cardiac area. You may be asked how you are to distinguish a pericardial from an endocardial sound. A well-developed to-and-fro friction-sound, such as I have just described, is unmistakably pericardial; so also is a well marked double aortic or double mitral murmur, with the loud blowing character, quite unmistakable; but sometimes at the base of the heart we hear a to-and-fro sound with characters intermediate between those of ordinary pericardial and endocardial sounds, and it is exceedingly difficult to say at once whether we have to deal with endocarditis or pericarditis, but in such developed cases, by carefully noting all the attendant circumstances, the extent of cardiac dulness, the position and character of the cardiac impulse, and the character of the pulse, we can with care usually come to a right conclusion. The great difficulty comes when we hear a faint adventitious sound during the early days of an attack of rheumatic fever; we have then these points to guide us. Pericardial sound is heard most commonly at the base of the heart, endocardial sound more commonly at the apex, the point of maximum intensity differs in the two; the area over which we hear the pericardial sound is at first very limited, whilst the endocardial sound spreads from the apex into the axilla. The endocardial sound is a low-pitched blowing sound, whilst there is usually a creaking character to be detected in the pericardial sound. It may, however, be impossible for two or three days to decide; the difficulty is increased by the fact that endocarditis and pericarditis very commonly co-exist at this stage of rheumatic fever.

Rheumatic pericarditis spreads all over the pericardium. In fatal cases we find that a great quantity of lymph has been thrown out, and if the inflammation has been very severe we find red blood corpuscles mixed with the exudation, and even massive hæmorrhage, but we never find pus. I shall devote a special lecture to pericarditis, and shall then detail to you in what conditions we find pus in the pericardium, and therefore all that I need say here is that for years and years I have been looking for pus in the pericardium in cases in which pericarditis has arisen in connection with rheumatic fever, and I have only found it in one instance. Pericarditis, like endocarditis, is accompanied by more or less inflammatory change in the muscle of the heart, and I have often demonstrated in the post-mortem room that in fatal cases of pericarditis and of endocarditis alike, death is due to the concomitant changes in the wall of the heart. The muscle looks soft, it is much more friable; under the microscope we find the connective substance of the heart charged with granular matter and leucocytes—this is what is called myocarditis.

I will tell you another experience we have had in cases of rheumatic fever. There may be no sign of endocarditis, there may be no sign of pericarditis, but the breathing is quickened and the heart sounds are curiously muffled, and the medical man says, "I do not know what is the matter with the heart. I cannot hear the heart-sounds clearly; but there is no extension of the dulness upwards to indicate that the muffling is due to a large effusion in the pericardium; there is no rub, and there is no murmur. Why is it that I do not hear the heart-sounds clearly, and that the patient has such a small pulse and is so exceedingly weak?" Now with such symptoms as those, suspect myocarditis.

Here is another fact bearing on this subject. A patient has had rheumatic fever, and has had no pericardial disease and no valvular disease, but after the fever the left ventricle becomes dilated and he dies of heart failure. I remember a lad who had a double bruit over the apex of his heart, and the heart was much dilated; I thought the case was one of mitral stenosis and regurgitation. When he died I found there had been no valvular disease, and the sound I had heard had been caused by a rough fibroid thickening of a small area of the pericardium just over the apex of the heart. The left ventricle was greatly dilated and this had killed him. In such cases we are led to think that the disease of the heart results from myocarditis, inflammation of the connective substance of the heart.

There may be muffling of the heart sounds with dulness much extended towards the top of the sternum, the dulness being pear-shaped or conical, with the base of the cone about on a level with the apex of the heart, and the apex of the cone near the top of the sternum. When we find that we know that there is a large collection of fluid in the pericardium. These cases are now comparatively rare; when bleeding was in fashion they were much more common; the reason for this seems to be that the tendency for the pouring out of much fluid only comes on when the patient is in an extremely asthenic condition.

The friction-sound returns as the fluid is absorbed and the dulness diminishes.

Here is a question of great practical importance. Is it common in the acute stages of rheumatic fever to find some increase in the area of cardiac dulness when there is no murmur and no pericardial friction? Yes, this is not uncommon. What is the significance of it? It means that the wall of the heart is weakened, that the contractility has failed to some extent. If this continues what are we to expect? That there will be an outburst of inflammation in the heart.

As the pericarditis, endocarditis, and associated myocarditis become more severe there is more and more difficulty of breathing, and we measure the severity of the heart's inflammation by the degree of dyspnœa.

We have to bear in mind that with the pericarditis and endocarditis there is very commonly pneumonia of the base of the left lung. Now I want to impress this on you, that if there are pericarditis, and endocarditis, and pleuritic friction, and signs of consolidation of the lung, with extreme dyspnœa, be careful, if it is in a primary attack of rheumatic fever, not to give a discouraging opinion, for the patients commonly recover. In a primary attack of rheumatic fever the patient may lie in bed for weeks with such extreme dyspnœa that it looks as if he would be suffocated in a few minutes, but he will most likely recover, and perhaps recover to such an extent that a few weeks afterwards we shall be unable to detect any sign of disease.

Before leaving this subject let me add that we must always look out for fluid in the pleural cavity, but generally it does not accumulate so much that it becomes necessary to draw it off. There is also very commonly some amount of bronchitis; but we rarely find in primary or secondary attacks that the bronchial tubes become choked to any great extent.

Whilst these disturbances are going on in the heart and in the breathing, there must necessarily be much disturbance in the cerebro-spinal system. There is much wakefulness, and one of our most anxious thoughts is, how much we have to put the patient to sleep—to what extent we can put the patient into a stupor by a narcotic and not do more harm than good. I will say this to you: err on the side of wakefulness, it is safer. If you are in doubt, if you do not see your way clearly, do not give narcotics, for delirium is safer than coma. Years ago in lecturing to the students I used to tell them, following Trousseau, that cases in which there are occasional periods of delirium, the patient having hallucinations, saying for instance that there is some one under the bed, usually terminated fatally. But in my experience since that time I have come to think this a mistake. I am no longer afraid of delirium, and I am therefore better able to help the patient. You will be much impressed to see how wonderfully quick a delirious mind is to find out what is in your feeling, it has the instinctive quickness that belongs to animal life. If therefore you are frightened by delirium, your patient will find it out and be intimidated; but if you stand by his side with confidence, and humour his delusion by saying something like this:—"Do not mind them when they come up at night and sit on the beds and say queer things, but tell them to sit as long as they like," then you will give the patient confidence and help him through his trouble. When breathing and oxidation are much embarrassed, the mind must alter accordingly—offer no resistance and the trouble passes away.

The delirium may take a much more acute form. It is busy, the patient talks about his business, is exceedingly wakeful, with much tremor and great desire to get out of bed. Such delirium has more the form of delirium tremens, and is commonly the result of previous excess in the use of alcohol. It is what, if there were a broken leg, the surgeons would term traumatic delirium. There may in other cases be a very transient delirium, rapidly giving place to stupor—there is a condition like that in apoplexy; in some cases the disturbance in the brain may be marked by recurring epileptiform convulsions. These conditions, which were formerly known as cerebral rheumatism, usually disappear as the healthy circulation of the brain is restored. In the earlier years of my practice these forms of brain disturbance were much more common than they are now; in those days all patients with rheumatic fever were bled as a matter of course, and hence there was greater liability to disorder of the cerebral nutrition.

I have to speak of one other form of delirium, but this usually comes on later, when the acute stage is over, and the patient is tending towards convalescence. This is maniacal delirium. There is some curious connection between insanity and rheumatic fever. Dr. Savage, for many years superintendent of Bethlem Asylum, has often put this question to me: "How do you account for this? A patient comes into the asylum insane; then he gets rheumatic fever, and the insanity disappears, but returns as soon as he recovers from the fever." Again and again has he asked me that question. I want to bring home to you that, in studying rheumatic fever, and in seeking guides for treatment, we have to bear in mind how powerfully the great nervous workings of the body are affected in this disease. I told you that rheumatic fever might be ushered in by inflammation attacking the seventh cranial nerve. It may also happen that the meninges of the cord are first attacked. The first sign that there is anything amiss is that paraplegia comes on, and in the course of a few days this may rapidly disappear and the joints become inflamed.

Now let us go on to consider the course of endocarditis. It is not uncommon for the signs of endocarditis to disappear; the signs may disappear soon after the primary illness, or it may be after they have persisted for some time, even for years. For this reason medical men are more willing than formerly to recommend patients with a mitral murmur to insurance offices.

Most commonly however endocarditis leaves some thickening of the valves. If the aortic valve is affected, it is secondary to mitral affection; we very rarely find that the aortic valve is alone affected by rheumatic endocarditis. The thickening affects the edge of the mitral valve; it commonly also affects the body of the valve, and extends more or less into the fibrous ring at the root of the valve, and in some cases this ring may be extremely thickened and contracted. The chordæ tendineæ of the valve are similarly thickened, and the thickening usually extends up into the aortic valve to some extent. If the aortic valve is much affected, the flaps are thickened and adherent, and so shortened that they can no longer meet. The thickening makes them rigid, hence they can no longer lie back against the wall of the aorta, for these reasons there is usually in such cases both aortic obstruction and regurgitation. If the mitral orifice is extremely contracted, there may be simply a præ-systolic murmur at the apex, and no systolic; if it is moderately thickened, there is usually a long systolic murmur, preceded by a short præ-systolic. If there is little or no contraction, but well-marked incompetence, there is a long systolic murmur.

Sometimes after rheumatic fever in which there has been an attack of pericarditis, we see a systolic recession of the chest-wall over the apex of the heart. What does this signify? It means that at the apex of the heart, over a small area, the parietal and visceral surfaces of the pericardium are adherent, and also that the inflammation has extended through the parietal layer, so that there is tough fibroid tissue connecting the outside of the pericardium with the tissues of the intercostal space. So that we can diagnose adherent pericardium? Yes; in these cases we can diagnose adherent pericardium, but we commonly pay no attention to it, for the diagnosis is practically useless. This recession at the apex is only seen in cases in which the adhesions are so limited that they do not lead to any ill effects. When the pericardium is adherent throughout we commonly fail to diagnose it. There is not then the recession at the apex that we see in cases of trifling adhesion, and the symptoms produced by serious adhesion of the pericardium are not such as enable us definitely to refer them to their cause.

Pericardial adhesions are either harmless, seeing that they produce little or no hindrance to the action of the heart; or in the course of time they lead to great dilatation, with more or less hypertrophy of the left ventricle. Now, here comes a very interesting question: How is it that in so many cases there has been adherent pericardium for many years, as we know from the date of the attack of rheumatic fever which led to it, and, even without a history, the firmness of the adhesions would tell us that they were of long standing, and yet the left ventricle has not been dilated; and, on the contrary, in other cases, in which the appearance of the adhesions is similar, there was fatal dilatation of the ventricle? How is it that the ventricle is dilated in some cases and not in others?

What do we understand by dilatation of the ventricle? Muscle is a most specialised elastic substance; it takes in so much energy; it contracts and gives it out again. It, in a sense, resembles a piece of india-rubber or a spring; we stretch these, and by their elasticity they contract again, and in so doing restore the energy we have used to stretch them. A healthy muscle works in strict equilibrium—taking in so much energy and giving forth the same, and so keeps its normal size and position. But in these cases the left ventricle has lost that power; it is, in a sense, like a piece of rubber that is getting worn out, and as it loses its elasticity it becomes more stretched without power of recovery. Next we have to ask what it is that gives the healthy muscle its power, its energy? The energy of muscle is dependent upon red blood.

Reed demonstrated that directly; he showed that the heart supplied with venous blood could only go on beating for a little while. I have shown you often how the heart's muscle has ceased to act because the blood has been cut off from it. And we know that death from anæmia is death from failure of the heart's muscle. Now, anæmia is one of the most marked symptoms of rheumatic fever; both before, during, and after the attack of fever there is anæmia.

We begin to see what are the conditions which lead to failure of the energy of the heart, to dilatation of the left ventricle. Loss of red corpuscles is one of the most important; but there are other things which lack of knowledge prevents us clearly enumerating. Insufficient supply of albuminous material starves the heart. Owing to failure of appetite, sufficient food is not taken, and the body wastes and the muscles waste. We have reason to think there is insufficient alkali and insufficient water. It is astounding to find what nonsensical ideas people have about water. A man came to see me the other day—a learned person, whose mind was crammed with books, but his body had wasted away—and he said to me, "I thought it was wrong to drink much water!" He used to see the railway engines working away, and yet he was afraid to drink much water! This is a common story.

Besides red blood-corpuscles, albuminous matter, water, heat, light, and electricity, the working of the heart is dependent on another great power, and that is nervous energy. I mean by nervous energy the power that works in the nervous tissues of animals—and of plants also, for we have been coming to see that in the vegetable world, as in the animal there is powerful nervous action.

Seclusion indoors and loneliness are most killing to the circulation; rheumatic fever and excessive indoor life go together. This is what I have been leading up to, for I have to account for the dilatation of the left ventricle. We may talk about what we see on the post-mortem table, and say that the dilatation of the ventricle has come from the growth of fibroid tissue in its wall. The patient is dead by that time, and is beyond our help; we want to know what are the living conditions which led to the growth of the fibroid tissue.

Healthy constitution of the blood was lacking, and there was failure of nervous energy: these I doubt not were the conditions that allowed the fibrous tissue to go on extending into the wall of the heart.

The practical conclusion we are led to is this, that after rheumatic fever, if you have reason to fear adhesion of the pericardium, be sure that the patient is a great deal out of doors, and that his movements are not restricted more than is absolutely necessary. Arms and legs know how to work with heart, but I do not. I often say to a patient, "Do not ask me how far you may go, but trust to your ease of feeling." All that I have been saying to you about thickening and adhesion of the pericardium applies equally when we have to counteract the thickening and contraction of the mitral valve and the mitral orifice, and when we want to prevent the thickening extending from the valves into the wall of the heart. After rheumatic fever the question before us is, how are we going to get the patient at work again, with as little damage to the heart as possible? We have proved that drugs are a failure. I know of no medical man who will not admit that drugs are powerless to remove the damage done to a damaged heart. Pathology teaches us that the great thing after rheumatic fever is to get the patient into the open air as soon as he is free from acute symptoms; get his limbs at work, and be sure that he has enough food, water, &c., to enable him to have healthy blood circulating in the vessels of his heart and brain.

Death very rarely occurs in a primary attack of rheumatic fever. Death is more common in a second attack, but still rare. Usually when death occurs in connection with acute rheumatic symptoms we learn that the patient had had rheumatic fever three, four, or more times previously; and we also think that each successive attack left increased injury to the heart. We are led to think this because we are told that each attack left the patient more short of breath and less strong, less capable of going on with his work. We see this when we watch a patient through several attacks of rheumatic fever, that he becomes progressively weaker and more breathless, and we notice a cachectic, pale appearance, denoting an increasing tendency to rheumatic anæmia. The patient finds it more and more difficult to get rest—always a most serious sign of increasing weakness—until his nights have become almost unbearable.

At this stage we notice that with each rheumatic attack the arthritis seems to be less pronounced, but the heart trouble becomes more and more severe, until at length the case has this aspect. There is very little affection of the joints, there has been nothing more than a little pain, redness, and swelling, in one or two of the joints, quite trifling, and yet the patient remains so weak; day by day his breath is becoming quicker and shorter, and it looks as if death were near at hand. We find that the temperature is raised day after day. Why is the breath so short and why does the fever continue? There is not anæmia sufficiently extreme to account for it, and there is no evidence of any extensive disease of the lungs or of the kidneys; what, then, is the cause of the continued shortness of breath, with raised temperature, with increasing weakness, while the joint affection is comparatively insignificant? The cause is endocarditis is smouldering on in the damaged valves, and the inflammation is involving the wall of the heart more and more. This is the way in which rheumatic fever commonly kills, when there have been several previous attacks.

Do not overlook the fact that in children suffering from rheumatic fever, the common experience is, that they are apt to have little joint affection, with much heart failure. Their joints have not been much used, are not so much worn, and therefore are not so liable to inflammation. Two opposed conditions are free from disease; when structures are perfectly healthy they do not tend to become inflamed, and when they are extremely worn they have no tendency to take on inflammatory action.

Whilst acute inflammatory action is going on in the endocardium, patients are liable to embolic conditions; these are very rare in primary attacks, less rare in secondary attacks, and become more common in later attacks. One day the patient may be seized with pain in the left side, and there is a sudden rise of temperature. On examination we find that the side is tender, and that the area of splenic dulness is enlarged; we make the patient take a deep breath, and we then feel the edge of the spleen below the costal margin. We then say at once that the spleen is enlarged, inflamed, and congested, and we infer that there is embolism of the spleen, and that the tenderness over the organ is due to a little local peritonitis. We may perhaps find some albumen in the urine, and this leads us to think that there may be renal embolism; and the existence of this condition is rendered still more probable if there is hæmaturia.

On another day we may find that the patient has a sudden cerebral disturbance, it may be simply a transient giddiness, in rare cases there may be a distinct epileptiform seizure; very commonly, in these cases, the disturbance takes the form of a sudden inability to speak; the patient can move, can make signs, and perhaps begins to laugh with astonishment, an hour later we find that he can speak just as well as before. I remember a case in which an attack of that kind was soon followed by a similar one, of rather longer duration, and there was a third and the loss of speech was permanent, and there was also loss of power over the right arm and leg. You will find many similar cases recorded.

With such embolic changes in the brain there is usually rise of temperature, and drowsiness tends to come on, passing into stupor, the breathing is quickened, and there are sordes on the teeth and lips. We conclude that there is red softening of the brain, producing hemiplegia.

What do we find on post-mortem examination in such a case? The embolic condition of the brain is most commonly in the area of distribution of the middle cerebral artery, and more commonly in the left hemisphere than in the right; it may, however, occur in any part of the brain, but is comparatively rare in the pons. I have found it most frequently either in the convolutions of the brain, or in the corpora striata or optic thalami. If the embolism leads to death in a very short time, an exceedingly rare event, we should expect to find simply an anæmic condition of the brain, death having occurred before there has been time for softening or inflammation to set in; but in my opinion this view is rather theoretical than practical. We usually find that the portion of the brain supplied by the plugged artery is extremely congested, that blood has been extravasated into the brain substance between the distended vessels, and that the affected area is of softer consistence than the normal. We are led to think that when the artery has been plugged the small vessels from which the blood has been cut off become paralysed, and that the blood flows back into them from the veins, so that they become extremely distended. Soon the stagnant blood penetrates the walls of the vessels, there is increasing softening of the affected area, and the piece of brain perishes, so that in extreme cases we find it of a dirty brownish-red colour, and in a sloughing condition. More or less inflammation, cerebritis, arises round the softened area, and if the patch is large, and is situated near to the medulla oblongata, in the optic thalamus for instance, the breathing power fails, and there is increasing coma, leading to death.

With this embolic condition of the brain there may be embolism of the kidneys, skin, spleen, and lungs; the patient may have repeated shivering attacks, increased temperature, sordes on the lips and teeth, a brown, parched tongue, and other pyæmic symptoms; the rigors are like those of pyæmia, the temperature is like that of pyæmia, the patient is in a typhoid state, and is evidently dying, and we may find a good deal of swelling of some of the joints. Now, this condition has been spoken of as a pyæmic morbid state due to endocarditis with embolism.

At the post-mortem examination in some of these cases we find changes in so many organs that this condition has been familiarly spoken of as "showers of emboli."

There is another condition connected with rheumatic fever to which I must call your attention, and that is chorea. What is the relation of chorea to rheumatic fever? Before we can answer that question we must understand clearly what we are to recognise as chorea. There are certain movements of the limbs, a certain expression of the face, and when we see them we say at once, "It is chorea;" the movements and the facial expression are absolutely characteristic. We next have to ask, seeing that these movements are merely an outward manifestation, whether they are always dependent on one kind of morbid change within? We have come to see that these choreic movements may have different internal origins; they may be due to senile changes in the brain; they may be connected with epilepsy; they may be connected with rheumatic fever: very commonly there is some indirect relation with rheumatic fever. The patient has perhaps not had any of the ordinary manifestations of rheumatic fever, but has mitral disease, which has come on insidiously, and then there is a choreic outburst. In other cases we find that the patient's brothers and sisters have had, some of them chorea, and some of them rheumatic fever. Or the patient may have rheumatic fever at one time and chorea at another. Lastly, the choreic attack may come on at the end of an attack of rheumatic fever. It is therefore beyond all question that there is some close connection between chorea and the rheumatic fever state.

The matter is so important that I will repeat the observations that have been made in this connection. The symptoms of chorea may precede the ordinary manifestations of rheumatic fever; they may come on during the decline of the rheumatic symptoms; the symptoms of chorea may alternate with rheumatic outbursts; they may be attended by sweating like that of rheumatic fever, without any joint affection.

Chorea usually begins with restlessness, with what is often termed fidgettiness, and with much mental excitement. After a time we notice the typical choreic movements in the face and hands; restless gyrating unequal movements; very commonly the tongue and other muscles concerned in articulation are affected, so that the speech becomes jerky and indistinct. If the attack is severe the movements affect also the lower extremities, and the body generally, and in the worst cases the movements are very violent, and continue day and night until the energy of the body is exhausted, and there is great danger of death from asthenia. We measure the risk by the force of the pulse. The loss of energy in the muscular system is generally in chorea, as in other diseases, accompanied by loss of energy of the heart, so that as the patient is tending more and more to become fatally exhausted we find the pulse gets softer and softer. With the choreic movements of other muscles there may be choreic agitation of the wall of the heart, leading to marked irregularity of rhythm. In severe chorea there is always great risk of endocarditis. If you are asked, What are the morbid changes you will certainly find in fatal cases of chorea? answer, "Acute endocarditis."

We always find recent acute endocarditis at the post-mortem examination on a case of chorea. If the valves have not been previously affected, we find a thin row of vegetations on the auricular surface of the mitral valve near the free edge; if, on the other hand, there has been antecedent rheumatic change in the valve, we find the acute endocarditis occurring amongst old fibroid thickening. But it has been most surprising, after death from chorea, in which the disturbances have been so extremely violent, to find no morbid appearances but a few vegetations on one of the valves of the heart.

The pathology of chorea has been a puzzle to us all. We have thought that with all these movements we might expect to find some change in the spinal cord, but there was nothing. We have examined the convolutions of the brain most carefully with the microscope—I have spent much time myself in cutting up brains—and we have found in some cases evidence of inflammatory exudation into the convolutions of the brain, but in other cases we have not been able to find this. Certainly the more carefully I have conducted my examination the more often have I found evidence of œdema and inflammatory exudation into the convolutions, so that it cannot be disputed that such a condition is often found after death from chorea. But this does not help us much, for we find the same conditions in other cases in which there has been no chorea. I remember a case of rheumatic fever in which death occurred with cerebral symptoms—so-called cerebral rheumatism—and in this case the morbid appearances in the brain were precisely similar to those which have been found in cases of chorea.

What are we to do in chorea? If, after a river had burst its banks, and flooded all the surrounding country, I were to go and look at the flood and say, "The bank has burst!" you would say, "Any fool can see that; but why did not they recognise the strength of the river long ago, and take measures to prevent its bursting its banks?" Now we are in very much that position with regard to chorea. In families with the rheumatic tendency we may watch chorea coming on; we may notice that a child has restless fidgetty ways, and that these gradually pass into choreic movements, and we must warn the parents that unless the child's nervous system is rested there will be an acute outburst of chorea. Now, when that comes, the energy of the body breaks out of its proper channel, like the water through the broken river-bank; and just as the river-bed in such circumstances may soon run dry, so in chorea, when death occurs, it is simply because all the energy of the body has been drained away. This view helps us to understand how it is that we find so little on the post-mortem table to account for death.

This brings me back to what I told you at first about rheumatic fever, that it may kill, and yet we may find little or nothing to account for death. In such cases the immediate cause of death has usually been hyperpyrexia. A patient with rheumatic fever may seem to be going on in the usual way, and then in a few hours the temperature runs up to 110°; as this occurs, there is increasing evidence of failure of the heart, the pulse becomes much smaller, lividity sets in, there is extreme restlessness, and the temperature remains up. In these circumstances you must have but one thought—to reduce the temperature. At all hazards put the patient into a cold bath to lessen the heat. I have done this and have been most encouraged to find that the pulse got stronger, that the lividity disappeared, and that the patient eventually recovered.

Bear this fact in mind, that when a person of an exceptionally sensitive nature is suffering from a severe feverish attack—a young and very restless person—an outburst of fever, causing hyperpyrexia, is liable to come on in a most surprising manner. What do I mean by saying, "in a most surprising manner?" I mean that the patient is suddenly found to be very near death, and that the medical man is sent for in a great hurry, and that when he comes he has but one idea, to get the temperature down very soon, or the patient will be dead.

You may be asked whether you think that rheumatic fever is a disease of the nervous system. Does not the erratic character of the arthritis suggest a disturbance in nervous function? The pain flits about from part to part in such a peculiar way; and do we not meet with similar attacks of arthritis in cases of undoubted disease of the spinal cord? Are not rheumatic patients usually very nervous people, and liable to other diseases of the nervous system, such as chorea? Well, some have favoured that view, that rheumatic fever is a disease of the nervous sytem; and this much is indisputable: that the nervous system in rheumatic fever is not in ease—that is to say, that it is diseased in the widest and oldest sense of the term. But we must remember this, that whatever views we accept in pathology, they are insufficient if they do not help us to cure the patient; it is of no use for us to lay flattering unction to our souls, to pride ourselves on our knowledge, if we cannot cure the patient. Now this view, that rheumatic fever is a disease of the nervous system, in no way helps us to clear views about the obscure beginnings of the disease; it does not explain the anæmia, the sour sweats, and the increasing weakness which are the forerunners of the arthritis.

Others have said that the excessively sour perspiration, the acidity of the saliva, and the extreme acidity of the urine, afford evidence that there is some acid poison in the system, and they hold that such acid poison is the cause of rheumatic fever. That was the accepted view of the pathology of rheumatic fever, and is held by many at the present day. So they said the remedy must be this: "Let us pour in a lot of alkali; let us give bicarbonate of potash every two hours, until the perspiration, the saliva, and the urine are alkaline." That was the prevailing treatment, admirably consistent with the theory, and the only unfortunate point about the matter was that the theory was wrong. It was found that the treatment with alkalies had no influence in preventing heart disease, and it was seen that the cases ran a certain course, uninfluenced by the treatment, some terminating in five or six days, some lasting many weeks. Moreover, it was recognised that the treatment tended to increase the anæmia and also the asthenia. But what chiefly led to the abandonment of the treatment was the generally admitted failure to arrest the heart disease. But perhaps I am wrong in saying it was the failure of the treatment that led to its abandonment. You will find that Buckle, in his "History of Civilisation," says that a practice is never abandoned, no matter how destructive, until the theory on which it is based has been changed, and as an instance of this he refers to bleeding, and to other long-disused methods of medical treatment. And I think the alkaline treatment of rheumatic fever is another instance of this truth. Of late years the theory has been generally given up. It has been recognised that the excessive acidity is a consequence of the rheumatic fever process, and not its cause. It has been recognised that these acid products are the products of tissue activity, and that the normal alkalinity of the blood and tissues is maintained by a proper relation between their formation and excretion ; and that excessive tissue-waste on the one hand, and diminished excretory activity on the other, will lead to hyper-acidity of the blood and tissues.

Now, I have been always impressed with one thing in the treatment of rheumatic patients, and that is, that we must promote excretion. In patients liable to rheumatism we must avoid much alcohol, confinement indoors, much food, and little exercise. They get worse under such treatment, and much excretory action is indispensable to them.

I think, after what I have told you, you will be disposed to accept the acid condition of the body in an attack of rheumatic fever, to accept it as you find it. What you must do is, try to promote easy rhythmical action of the heart by rest. To facilitate breathing, and so help the right side of the heart, let the patient have plenty of fresh air. You will let delirium vent itself before pushing the patient too much into stupor by opiates; you will give a sufficient amount of food for freshness and strength, but not enough to clog.

In our present state of ignorance, it is impossible to speak clearly about the causal conditions of rheumatic fever. We have, however, come to this conclusion, that rheumatic fever, nervous affections, and phthisis are allied morbid conditions. In a family, one member may have rheumatic fever, another phthisis, and another hysteria or some other nervous affection.

LECTURE XIII.

RHEUMATIC GOUT.

In one of the standard works on chronic rheumatic arthritis, which is rheumatic gout, you will find the following statement: " It begins with rheumatic fever." This work is recognised as being very carefully written by a man who has seen a great deal of this disease, but nevertheless his statement that the disease begins with rheumatic fever is not true. I should rather say that it is at once true and not true. At the time that was written it had not been clearly recognised that there may be arthritis in several joints, very like rheumatic fever, but the whole attack differing from rheumatic fever in this, that there is little or no liability to heart affection. In certain patients we find a liability to arthritis in several joints, with high temperature, somewhat sour sweat, furred tongue, and the case looks very like an ordinary case of rheumatic fever, but the patient does not get endocarditis or pericarditis. Now how are we to distinguish such cases of arthritis from ordinary cases of rheumatic fever with liability to heart affection? The arthritis is not so erratic, for it occurs in a joint in which there is already some chronic degenerative change, and the inflammation tends to linger in the joint for days, and even weeks; in this we have a most marked contrast to rheumatic fever, in which the inflammation flits about from joint to joint. Further, the temperature is not so high, the sweat is not so sour, and after a few days' illness we find the heart is still unaffected. Under these circumstances we may be confident that we have not to do with rheumatic fever, but with acute arthritis connected with rheumatic gout.

These cases are generally more tedious than cases of rheumatic fever, they extend over weeks, the arthritis recurs and recurs, and the patient may be laid up even for months, but during the whole of that time we find no evidence of endocarditis or pericarditis. You will naturally ask me if there is no exception to what I have just said—have you ever known a rule with no exception? The only exception is this, that if there have been antecedent atheromatous and calcareous disease of the aortic valve we may get acute endocarditis arising in the damaged valve. Acute or sub-acute arthritis, such as I have just described, may occur in young people, and then it may be some days before we can be sure that the case is not one of ordinary rheumatic fever. An acute attack of rheumatic arthritis is liable to be followed months afterwards by phthisis. When I was physician at Victoria Park Hospital the phthisical patients used often to say they had suffered from rheumatic fever; now in these cases there was no evidence of heart disease, and I have no doubt it had been rheumatic arthritis of the kind I have been speaking about. Children also are liable to chronic changes of a rheumatic gouty kind in the joints, and from time to time there may be attacks of acute arthritis; I have known this occur as early as eight years of age.

Now let us ask, What are the characteristic changes in the joints in what has been variously called rheumatic gout, chronic rheumatic arthritis, arthritis deformans, and osteo-arthritis? One of the first things that we notice is a fibroid degeneration in the articular cartilage; it loses its hyaline appearance and becomes marked by lines or is fibrillated. It is evident that the cartilage has a tendency to perish; we notice further that it wears away and becomes thinner and thinner at the parts of greatest friction. The fibroid condition tends to pass into ossification. In most cases there is also over-growth; this may occur in any part, but is most marked along the outer edge of the articulation. Here it commonly spreads round the joint, so that there is a projecting ledge around the articular surface—the so-called lipping. The outgrowths on the opposed articular surfaces may interlock, so as to cause spurious anchylosis.

While these changes are in progress the atrophy and rubbing away of the cartilage continue, so that the bone is laid bare. The movement of the two bony surfaces over one another makes them very smooth and hard ; they look like pieces of ivory, and the condition is known as ivory eburnation. The roughness and ossification of the cartilage and the denudation of the bone cause a grating in the joint when it is moved.

While those processes are taking place, there is more or less fibroid degeneration in the connective tissue around the joint, leading to thickening of the capsule ; but the thickened ligaments are weaker than healthy ones. For this reason, that the ligaments are so weak, partial dislocation is not uncommon. What the patients chiefly complain of is the pain in the joints, the creaking when they are moved, and that they give way sometimes when they are used. Bony outgrowths around the intervertebral articulations are very common in rheumatic gout, and the patients often come complaining of the cracking in the neck caused by such outgrowths.

With these extensive degenerative changes you will understand that the joints are exceedingly liable to inflammation, and that the inflammation is prone to linger in the joints for weeks and months. Cases in which there is much synovitis lead to effusion into the joints. The cases of dropsy of the knee so common in surgical practice, lasting for weeks or months, are of this nature. As the changes in the joints are chronic and progressive the arthritis is exceedingly liable to recur repeatedly. In some cases the pressure of fluid in the joint becomes so great that we have reason to apprehend destruction of the cartilage, and sometimes in cases of extreme distension acute inflammation arises, and the joint is completely destroyed. I have already told you that these degenerative changes in the joints may occur in adolescence, or even in childhood, but they are most common in people from forty to sixty years of age. At that period of life they are exceedingly common, so common that I have been led to ask myself whether every one who lived long enough had not these changes in his joints in more or less degree. Are not these changes due to wear and tear of the joints spread over many years, and not usually manifested until at or after the middle period of life? Now that this is so is beyond question. We meet with these changes in association with emphysema, with atheromatous changes in the aorta, sometimes with granular kidney, or with chronic degenerative changes in the central nervous system. So that when we think that the patient is getting rid of the arthritis and is going to get well, he is not uncommonly attacked with pneumonia or broncho-pneumonia; then, as he is getting through that, some curious cerebro-spinal disturbance comes on. Sometimes the attack takes another form, it begins with sciatica, and after we have been treating the patient for weeks, rheumatic gout comes on in the joints. Again, I have known chronic rheumatic inflammation extend along the shaft of the tibia, leading to bony outgrowths, and I have also met with it in the spine, in association with intercostal neuralgia. In other cases the fifth cranial nerve may suffer. Another symptom that sometimes accompanies rheumatic gout is great failure of general nutrition. Some of the most extreme cases of emaciation I have met with have been in this connection. What is called muscular rheumatism may be the first symptom; the fascia of the lumbar muscles, or of some other group of muscles, suffers first. In other cases there is cachexia; the patients become pale, not exactly anæmic, but of a faint brown-yellow colour, and they get weaker and weaker, with a tendency to gradual failure of the heart. You will therefore not be surprised to meet with rheumatic gout in association with various diseases. It may be associated with insanity or with paraplegia.

LECTURE XIV.

GOUT.

There is one joint of the body that commonly more than all others is exposed to wear and tear, and that is, the metatarso-phalangeal joint of the great toe, for it is a small joint, and yet is subjected to extreme pressure. You know how often on the post-mortem table I have cut into this joint to show you the changes characteristic of rheumatic gout. You will be shown such a joint, and asked how you can distinguish the changes from those of gout. How do we recognise a gouty joint on the post-mortem table? We do so by the presence of lithates; there is a white, powdery deposit in the articular cartilage, and in the capsule of the joint. We have, however, to remember that the gouty and rheumatic gouty changes not uncommonly co-exist. Clinically there is a clear distinction between an attack of gout and one of rheumatic gout. Cases due to deposit of lithate of soda in the joints are characterised by severe pain coming on paroxysmally, setting in at about five in the evening, a burning, wrenching, screwing pain, which lasts till the small hours of the morning, and then subsides, to return at the same hour the next evening, and so on for several days. In rheumatic gout, while there may be some nocturnal pain, it is much more present in the day; in some cases it persists day and night.

Gouty arthritis—that is, arthritis in connection with lithate of soda deposits in the joints—usually comes on at about the same period of life as rheumatic gout, and in connection with the same kind of degenerative changes in the tissues, such as emphysema, aortic atheroma, granular kidney, degenerative changes in the central nervous system, &c.—these facts add to the difficulty of diagnosis. We are often guided as to the gouty nature of the illness by finding lithate of soda deposit in the cartilage of the ear, and further by hearing that the attacks have recurred again and again in the great toe; on examining the toe-joint in a case of long duration we find the chalk-like substance plainly visible beneath the skin around the joint. A similarly evident deposit may be formed around other joints, and old writers speak of patients who could score on the whist-table with the deposit in their knuckles.

Some gouty subjects are liable to attacks of arthritis when they are comparatively young; the attack lasts for two or three days, and then the patient seems quite well again, and there is little or no sign of degeneration of the tissues. These cases in which gout begins so early are generally due to inheritance.

Attacks of acute gouty arthritis may recur now and then over years, and the tissues still seem to be fairly healthy, but there comes a time when the attacks are more protracted. It is no longer a severe outburst lasting for two or three days, after which the patient feels better than he did before; the attacks are perhaps less severe, but they are more protracted, and during the attack we find some albumen in the urine, which disappears after the attack is over. We further notice the signs of emphysema increasing slowly year after year.

Now, what are we to apprehend in such patients? Up to that time the disease has been spoken of as gout, all the suffering has been attributed to gout. Now we find that the gout is more persistent, and it does not yield to remedies as it did in early days. The patient has ceased to have any confidence in remedies; he says that his gout is less severe, but that he seems hardly ever free from it. There are no severe outbursts now, but frequent twinges of pain.

The patient becomes weaker and weaker, and degneration of the tissues progresses. Such patients are very liable to dilatation of the left ventricle and to extensive degeneration of the vascular system, so that towards the end of such cases we commonly have to deal with the ordinary symptoms of dilated left ventricle and granular kidney, with subacute attacks of gout. Life is terminated in many of these cases by an epileptiform or apoplectiform seizure, or some other cerebral outburst.

In advanced gouty states the mucous membranes become very weak, and the gout may attack the throat, causing gouty tonsillitis. The patient is further liable to what is known as gout in the stomach. How are we to recognise the nature of such an attack? We are told that the patient has had attacks of gout for many years, and that latterly he has become very weak. He has perhaps had lately more or less suffering in his stomach, but now he has been rather suddenly seized with a burning, gnawing pain in the pit of the stomach, so severe that his pulse becomes small, his skin cold, and he looks as if he were going to die of collapse. There have been no signs pointing to ulcer of the stomach, and there is no evidence that he has swallowed poison. Vomiting comes on, his breathing is labouring, and we say at once that he has a severe attack of stomach disease, tending to rapid death from collapse. From the previous history we know that it is gout in the stomach. In one case at the post-mortem examination I thought I could see lithate of soda deposit distinctly in the wall of the stomach. There is a gouty deposit in the stomach very similar to that seen in the tonsils.

It is well recognised that there is such a thing as gouty kidney. What do we understand by a gouty kidney? It is a granular contracted kidney, with a lithate of soda deposit in it, usually in the pyramids. The lithate of soda, which the kidney is endeavouring to excrete, irritates its tissue, so that a chronic degenerative process is set up, and there is also a liability to attacks of acute nephritis. As the changes in the kidneys progress we find that there is persistent albuminuria, and other symptoms of chronic kidney disease—œdema, shortness of breath, &c. Do not overlook that there are also changes in the cerebro-spinal system in chronic gout, curious changes in the nervous system, leading to gouty angina, gouty asthenia, gouty gastralgia, &c.

I want you clearly to understand that gout is met with clinically in two distinct classes of cases. First, we have cases of gouty arthritis in which there is no appreciable tissue degeneration; such attacks may recur over years before there is appreciable tissue degeneration, and in such cases there is usually a clear gouty inheritance, though the patient may by his habits contribute to the development of the disease. Secondly, we meet with cases of gout in which there is evidence of well-marked degeneration of tissue. We have also to remember that when attacks of gouty arthritis have recurred for many years, degeneration of tissue usually supervenes. I used to see a very active and strongly built Scotchman, the subject of inherited gout, and he suffered from attacks of arthritis in his great toe. When he had gout and sent for me, he would say, "Give me a drachm of colchicum and an ounce of castor oil, for I must be at work again the day after to-morrow." He made me do that on more than one occasion—he bathed his foot in whisky and swallowed colchicum. One day I got so disgusted that I told him I would not see him again, and he thought I was a great fool for losing the money. I did not see him for two or three years after this, and then he had dilated heart, and emphysema, and albuminuria, and in a very few months he was dead. I was much impressed by this case that these tissue degenerations may come on very rapidly when the body is weakened by any cause. In his case his business affairs were getting complicated, and he had these frequent attacks of arthritis, he became asthenic, and his tissues quickly underwent degeneration. Our aim in treating gout is to relieve pain at as little cost as possible, and to endeavour to ward off degeneration of tissue. Colchicum and purgatives for acute gout are tending to go out of use, for the patient gets relief at too great a cost.

I once had a patient who had been a vestryman; he had had gout several times, and then had hemiplegia. I asked him, "What has been your experience with gout and port wine?" He answered that when he left off port wine the gout was better, but the man was worse.

People have come to learn that they may have worse things than an attack of gout, and indeed many patients long for the attack because they know they will feel better after they have had it. They feel better, stronger, able to eat with more relish, have calmer nights, and lose a dreadful depressing feeling of languor they had before the attack; this is a very old experience.

I have had this question put to me by a patient, "Do you think that I am suffering from suppressed gout?" The patient has noticed that he is better after he has had an attack of gout, and that he is very languid and depressed before the attack comes on, and he has heard doctors speak of suppressed gout, and so some day when he is feeling very ill he comes and asks that question. Now let us ask ourselves why it is that patients are so languid before an attack. At this period patients usually become very irritable, there is much gloomy feeling, the bowels are constipated, the urine is loaded with phosphates, the face is paler than usual, nights are disturbed, and the usual relish for things is lost.

The attack of gout begins with pain in the joints, and as the pain increases the joints become red, and then they swell. I repeat that the pain and redness are most marked before the swelling—before, that is, the vessels are relieved by the outpouring of serum. The urine is scanty and high-coloured, and deposits lithates, and the skin begins to sweat. After a time, when the pain subsides, the patient begins to sleep more refreshingly, more calmly, the gloom has gone, and his bowels act naturally, or he may be well purged, and the œdema passes away; in the course of a few days the urine becomes of a natural colour, the tongue clears, and the patient seems to have come out of a pit of misery. Now this is a common experience, and we cannot therefore wonder that patients should sometimes ask us, "Do you think that I am suffering from suppressed gout?"

I will give you another example. A patient is in an asylum with suicidal mania; the doctor finds it very difficult to prevent him from killing himself. One morning when the doctor is going round the wards, the patient points to his toe and says, "I am all right now, doctor:" he has an attack of gout, and his suicidal tendency is gone.

I will give you a case that occurred in my own experience. A lady was brought to see me from Devonshire, and she said, "Several of my friends have committed suicide, and the misery of my life is a feeling that I want to kill myself." She was a middle-aged woman, and as far as money was concerned she had more than enough. I saw her a year afterwards and she wanted me to publish her case in the medical papers. After she had seen me, she said, she was treated for suppressed gout, and the desire for suicide disappeared, and she attributed her recovery to the treatment for suppressed gout.

A patient is seized with a racking pain in the region of the heart, and the pain extends across the chest; he has a miserable worrying oppressed feeling; he dreads that he is going to die, his heart aches so. It may be the pain is so severe that we have no hesitation in calling it angina pectoris, and yet, we say, it is not quite the ordinary form of angina; there is not the same amount of sweating, there is not the full agony that we see in true angina pectoris. This pain may recur and recur for some days or more, and then there comes an attack of gout in the great toe or some other joint, and simultaneously the cardiac pain disappears. The patient has had gouty angina. Can you wonder if that man says that the pain at his heart was due to suppressed gout?

I will give you another illustration. A man is seized during the night with an attack of asthma. He has some shortness of breath before going to bed, and it gets worse during the night; throughout the day the breathing is better, but we hear some bronchial râles; at night the asthmatic seizure returns. In a day or two an attack of gout comes on and the lung trouble disappears. Now that is gouty bronchitis.

I might give you very numerous experiences of that kind. There may be toothache, sciatica, or lumbago; intercostal, facial, or other form of neuralgia; there may be iritis, or conjunctivitis; there may be tonsillitis, or trouble in the stomach; there may be eczema or urticaria, this last being very common; any one of these things may herald an attack of gout.

Now how are these things to be accounted for? Well there we will come to a full stop for a time, and turn to ask what are the antecedents of gout.

It has long been recognised that those who have an hereditary tendency to gout commonly suffer with constipation; that their urinary functions are liable to disorder; they commonly suffer with gravel, it may be lithate of soda, uric acid, or oxalate of lime; their skin is too dry; there is a weakness in the venous circulation, showing itself in the way they suffer from cold hands and feet; and they are very subject to colds, and to mucous discharges; there is weakness in the excretory functions of the body. We must keep these facts before our eyes, for young persons who have inherited tendency to gout will often ask us whether a tonic would do them good. Well, the tonic for them is sulphate of magnesia. Under some circumstances tartar emetic is a tonic. If a man has eaten too much and has a miserably overloaded stomach, with cramp in it, give him tartar emetic and make him vomit; that will act as a tonic for him. Now this very much applies to these cases.

With the weakness of excretory function is commonly associated a fine sensibility. Our male patients have a keen relish for the society of women; they enjoy their food and eat too much; they enjoy their wine and drink too much; the body feels enjoyment so keenly that it lives a little too fast. You may take this as a certainty, that whatever blessing has been given to mankind, men have learned how to abuse it. Let them abuse it, and be whipped until they come to use it properly.

We see then that there are several associated conditions tending to cause an excessive accumulation of dead material in the body; there is defective excretory function; and there is excess of food, both tending to cause accumulation of urates in the body. These people take little exercise and need little food, but they eat a great deal; the excessive use of alcohol interferes with the work of digestion and assimilation, and so the tendency to the accumulation of dead material is increased. If you study Greek history you will find that the Greeks suffered a good deal from gout, and that they attributed it to women and wine.

Now let us consider what an attack of gout does for the patient. It secures rest to the body, for the patient cannot walk about, and heart and breathing can go on more naturally; there is a little fever, and with the rest in bed the skin acts better; there is more temperate dieting, and the promotion of excretion by aperients and diuretics, and naturally the patient feels better after it is over.

Let us recognise that in suppressed gout there is a mine of suffering that must be relieved, and let us not overlook the fact that the subjects of it have sensitive natures, and that there is very commonly a tendency to insanity in the family stock, and we must do all we can to get them away from the idea of insanity.

I have often talked this question of suppressed gout over with men of large experience, and we have agreed that there are two things to be done: to get the mind into greater confidence by some impression on the feelings, and to get more freshness by increased excretory action.

Before leaving the subject of gout, there is another feature that I must put before you. As the body gets weaker, gout may come on in a form so acute and so general, that we may mistake it for rheumatic fever. It attacks many joints; the knees, ankles, wrists, one joint after another, is rapidly affected; there is a high temperature, sour sweat, and symptoms very similar to those of rheumatic fever. But if such an attack occurs in middle age or late in life, we immediately ask if it is not either rheumatic gout or true gout. At this age rheumatic fever is comparatively rare; we learn that there have been previous attacks of inflammation in the great toe, and that there is no heart disease; on the other hand, we are impressed by the amount of effusion into the knee-joints, and we say that this rather resembles rheumatic gout than true gout. We have to recognise that such a state of things comes on in asthenic conditions, and we must be careful not to purge the patients too much. They are very likely to die of broncho-pneumonia in emphysematous lungs, or of some acute disturbance in their degenerated brains.

When you put together all that I have said to you, you will come to this conclusion, that conditions that hinder excretory function predispose to gout. Years before distinct gouty symptoms arise there may be gravel or some other symptoms, which indicate that gout is likely to arise in later years, whenever there is a determining cause. Easy working of the nervous system, healthy venous circulation, and healthy excretion go on together; you will find frequent illustration of that. There comes an order of events which is termed worry (you will remember that worry in business or in other ways has long been considered a cause of gout), and then one day something occurs to put the patient into a violent passion, and immediately afterwards he has his first attack of gout. This onset of an attack of gout after a fit of temper has often been observed, as if the gouty poison has been slowly accumulating, and some determining cause were needed to bring about an attack of gout. The determining cause may be a drinking bout, or one of various other causes suddenly interfering with excretion.

If the nervous system is overtaxed there is insufficient oxidation. There has been too much office work, or too much study, and much food has been taken with a craving appetite, with the hope of keeping up strength, but there has been insufficient exercise, and not enough oxidation. Then the patient catches cold, or takes a long walk, something occurs which still further depresses the circulation, and gouty inflammation comes on.

Bearing all these things in mind, you will not be surprised to learn that women, who live comparatively protected lives, free from the worry caused by the competition for money and position, are not liable to gout until after menstruation ceases; this is a very old experience. The bearing of this is the influence of pregnancy.

Pregnancy is a most useful way of Nature; it keeps the heart and kidneys working naturally. When a woman feels that she has a baby in her womb, it makes her content with her lot. There may be a number of worries outside, but she is comfortable within. I have seen that so clearly, that sometimes I have said, "I hope that she will become pregnant," because I have seen, when we have to try and prevent disease, it may be gout, phthisis, insanity, or other, the wonderful influence of pregnancy. The pregnant woman leads a more healthy life, her mind's direction is more healthy, there is a deep order of love that leads to health, because she is building up. But when menstruation is past, and pregnancy is no longer possible, many a woman does not know what to turn to.

Then you will find that she is much indoors, often drinking to excess, with a miserable kind of diet, and a wretched way of life. I tell you these things, because we have got to find a remedy, because we have to guide the poor woman through the storm. But you will easily understand the truth of the old experience, that in women we rarely meet with gout until after the menopause, and you will understand the reason for it.

LECTURE XV.

GONORRHŒAL RHEUMATISM.

I KNOW nothing about gonorrhœal rheumatism from post-mortem examination, for it is never fatal—at least I have never met with a fatal case. What we know about this disease is from the actual suffering.

Clinically, we find that gonorrhœal rheumatism may take an acute or a chronic form. I have known it to be so acute that the difficulty was to distinguish it from rheumatic fever. The late Dr. Fuller, one of the physicians at St. George's Hospital, used to say that a mistake might easily be made between rheumatic fever and gonorrhœal rheumatism. When Dr. Gull and I wrote a paper about rheumatic fever, one of the objections urged by Dr. Fuller against the conclusions to which we came was, the possibility that some of our cases had been gonorrhœal rheumatism. He was a man of large experience, and I can fully endorse his views as to the possibility of confusing the two diseases. The onset may be very acute, with pain, redness and swelling of many joints, both in the upper and lower extremities. Some time ago I was reading Niemeyer on this subject, and found that he said the inflammation in gonorrhœal rheumatism never attacks the joints of the upper extremities. That is not true; if he had said the inflammation is usually in the joints of the lower extremities, and rarely in those of the upper, I should have agreed with him entirely. In these acute cases there is profuse sweating, a temperature of 102° or more, urine loaded with lithates, tongue furred, and, as I have said, they look very like patients suffering from rheumatic fever, but there is no heart complication. I once had a strong fireman lying in the hospital in such a condition that at first I thought it was a case of rheumatic fever, but when I observed the large amount of effusion into his knees I suspected it was gonorrhœal rheumatism. In these acute attacks of gonorrhœal rheumatism there is much effusion into the joints. The inflammation is not erratic as in rheumatic fever, but remains in the same joints for days. We then wonder whether it is rheumatic gout or gonorrhœal rheumatism, and we find that there is a gleety discharge, and that there is no lipping of the bones about the affected joints, and putting all these things together we come to the conclusion that it is gonorrhœal rheumatism. Such an attack may last for a week or ten days, and then the symptoms subside.

There is a subacute form, with very similar symptoms, but of much less intensity, and in such cases the difficulty is to decide whether it is gonorrhœal rheumatism or rheumatic gout. There is a subacute arthritis going on week by week; the patient has had gonorrhœa, and has still a gleety discharge, or has had one until quite recently; but there is a little lipping about the joints, and we are unable to say definitely as to how far it is rheumatic gout, and how far gonorrhœal rheumatism. I have been called in consultation to settle that, and could have no doubt that both diseases were combined. There was a man admitted to the hospital with gonorrhœal rheumatism. He lay in George Ward, and week after week, month after month, the disease went on. The effusion in the knee-joints persisted, and the aspirator was used again and again; then his joints began to thicken with bony outgrowths, and we were obliged to say that the case was one of gonorrhœal rheumatism, followed by rheumatic gouty changes in the joints.

There is also a chronic form. I used to recognise that in the out-patients the moment they came into the room. A man would walk in stiffly and gingerly, stiff about his legs, and tender under his heels. The disease comes on with tenderness about the tarsal bones, aching in the sole of the foot and round the heel, and it is exceedingly tender when the foot is put to the ground; this goes on for weeks or months, getting better now and then, but recurring again and again; there may also be a little swelling of the ankles, and perhaps of the knees. Seeing that the trouble is confined to the lower extremities we immediately ask the patient if he has had gonorrhœa, and we usually learn that he has had more than one attack of gonorrhœa, and has long been troubled with a gleety discharge.

It has been found very difficult to determine what is the connection between the gonorrhœa and the morbid change in the joints in these cases of gonorrhœal rheumatism. The gonorrhœa usually subsides before the arthritis comes on, and may have disappeared months before. We think that the inflammation is produced by some morbid change in the lymphatics, which travels down from the urethra until it reaches the joints, but I cannot tell you that this has ever been demonstrated.

LECTURE XVI.

PYÆMIA.

PYÆMIA may begin with arthritis, which is extremely difficult to distinguish from that of rheumatic fever, so difficult that I once passed through the following experience. I was asked to see a servant-girl, and I said she had acute rheumatism affecting her shoulder-joint, and that there was no danger. I was asked to see her again, forty-eight hours later, and found her bathed in perspiration, trembling, and nearly pulseless, and she died in a few hours. I was utterly astounded, and asked for a post-mortem examination. I found pus in the shoulder-joint, and no sign of rheumatic fever. Since that time I have had more than one similar case in the hospital.

The most characteristic feature of a pyæmic arthritis is the amount of œdema and swelling. Extreme swelling is not confined to pyæmia; we may see it in gout, in rheumatic gout, and in gonorrhœal rheumatism; but a very great amount of swelling and diffuse œdema about a joint is more characteristic of pyæmia. The joint is red, hot, and painful; day by day the swelling and œdema increase; fluctuation becomes distinct, and the skin over the joint shines because it is stretched so tightly; then we know that there is pus. We open the joint, and find that it is full of pus. Suppuration may extend from joint to joint, involving several of them, so that the patient is in bed for many months, with recurring suppuration in joint after joint, and becomes very much wasted by the protracted fever; his circulation is much weakened; his breathing is quickened and more or less difficult; he has occasional diarrhœa; there is much cachexia, a pale yellow colour, a beefy tongue; and yet from such a condition the patient may recover, after he has been ill for many months.

In searching carefully to discover the cause of such cases of pyæmia, I have found in many cases that there have been antecedent excessive fatigue, depressing circumstances, or great hardships. Excessive fatigue I regard as a kind of living death ; and in this condition the circulation gets weaker and weaker. Over-fatigue causes an excessive accumulation of dead material, and what will happen in such a state of things as that ? It is very like what happens in vegetable organisms when forced too much. What happens ? They contain much dead material, and various germs come and accumulate in them and destroy them. And so we should expect that in a joint much overworked, excessively worn, with perishing cartilage, bacilli would have an opportunity of accumulating. We find pyæmic pus charged with bacilli and bacteria.

As I may not again have an opportunity of speaking to you on this subject, let me here mention that the difficulty is to account for the presence of bacteria in health ; it is easy enough to account for their presence in diseased conditions, but that they should be found in health is a puzzle. In my own mind, I am not surprised at it, for I have been led to recognise that all healthy nutrition is accompanied by antagonistic action, by things that are ready to pull it down unless it keeps healthy. Such antagonistic action ensures the survival of the fittest.

We are therefore not surprised to find that pyæmia occurs where there is an enormous accumulation of dead material. If a hospital becomes overcrowded with a great many surgical cases, with much suppuration going on, with dead material accumulating in many ways, so that the air is poisoned, then erysipelas, infective cellulitis, and pyæmia become prevalent.

There is a condition more poisonous still than pyæmia, in which the patient dies before there is any evidence of local suppuration, and in which on post-mortem examination we find little or no local suppuration. This is septicæmia. Now in certain cases of pyæmia, on post-mortem examination, we find very little suppuration, it may be only one or two small abscesses of about half an inch in diameter in the lung, and yet the patient has perished; in other cases there is an enormous amount of suppuration, and yet perhaps the patient recovers. So that it is obvious that it is not the amount of suppuration that kills the patient.

You may be asked whether it is possible for a patient to recover after he has had repeated pyæmic abscesses in the lung. There is such a case on record. There was a man in Guy's Hospital who was watched for months and months, with extensive lung mischief, and he recovered and left the hospital. Later, he came into the hospital and died. At the post-mortem examination there was evidence of widely scattered pyæmic changes.

We come to this question, "What is the deadliest condition in pyæmia?" You may say bacilli, but that is not an answer that helps us much. We live by blood, and how is it that the patient comes to lose his life in the blood? No one has yet proved that the primary cause is the presence of so many germs in the blood. We want a view of pyæmia which may help us to get the patient through the disease.

What then are the antecedents of pyæmia? I have mentioned an enormous accumulation of dead material around the patient, and therefore pyæmia is common in hospitals, in military camps, and the like. Secondly, there is some antecedent condition of body that deadens the patient's blood and tissues, some antecedent cachexia. This may be due to great abuse of alcohol, or to excessive fatigue, or to close confinement in unhealthy places, places in which the blood has a tendency to perish rapidly when any determining cause is superadded.

We have to consider these things, in order to understand the origin of certain cases of pyæmia. For instance, a man gets a gumboil, and dies of pyæmia. Well, we say, that is a very extraordinary occurrence, thousands of people get gumboils and do not die of pyæmia. I have known a man die of pyæmia after a wound in the buttock. And yet we see other patients lying in bed for weeks with suppurating stumps, and enormous wounds, and they do not get pyæmia. I once made a post-mortem examination on the body of a man who had been lifting a piece of meat with a hook, when the hook ran into his finger, and he died of pyæmia. He seemed a well-nourished muscular man, and I could find no evidence of antecedent disease. All I can say is that I have generally looked upon such a patient as diseased, or a simple accident like this could not have led to these results.

Is there any form of cachexia associated with pyæmia? Yes, there is a well-marked pyæmic cachexia, a yellow colour of skin, with a peculiar dead smell which is known as the pyæmic odour.

Pyæmia is most commonly preceded by suppuration in some part of the body, and often the pus has decomposed. Very commonly the seat of primary suppuration is bone, but it may be in any organ or tissue, and it is supposed that from this primary seat the poison is carried into the general circulation.

I will give you an example of the way in which the infection may spread through the veins. The stump of a leg had suppurated after an amputation, and I found the femoral vein plugged with clot, but this clot had suppurated, and was charged with dirty yellow, unhealthy-looking pus. A portion of this clot had become detached, and I found it in one of the branches of the pulmonary artery; it had caused pyæmic abscess in the lung and pyæmic empyema, which had killed the patient.

At the onset of general pyæmia the patient is usually suddenly attacked with rigors. At that moment we suppose that the virus has become arrested in the lung or other part, most commonly it is the lung; rapid congestion has taken place, giving rise to great tension, and the disturbance of the nervous system so produced manifests itself in the form of a rigor. It seems that such a fresh lodgment of the virus may occur repeatedly, for rigors may occur again and again for many days. In the rigors there is rise of temperature and profuse sweating, and this leads to increasing cachexia, to increasing weakness of circulation.

If we listen to the lungs we may hear nothing but harsh breathing. Never be surprised if after these repeated rigors you merely detect harsh breathing, or only a few bronchial râles, for the pyæmic changes are confined to such a small area of the lung. But pyæmic changes in the lung occur near the surface, and so often give rise to pleurisy. If therefore in a case in which there have been repeated rigors pleurisy comes on, we say at once that there are pyæmic abscesses in the lung, with a secondary pleurisy. But in most cases there are hardly any physical signs of changes in the lungs—the circulation fails more and more, but we can only detect harsh breathing or a few bronchial râles.

What does a post-mortem examination reveal in a case of pyæmia? As you would gather, from what I said above, in the severest cases of pyæmia we find the slightest morbid appearances, and the pyæmic changes are always most marked on the lower parts of the lungs near to the surface. Our attention is usually drawn to their seat by finding some lymph on the pleura over the base of the lung, and we notice that this lymph is not a simple grey fibrinous exudation, but is purulent lymph, and underneath the purulent lymph there is a small patch of consolidation.

In the most rapid cases of pyæmia that patch of consolidation looks like a hæmorrhagic infiltration, it is of a blackish red colour like blood-clot, and we might take it for a small, abruptly defined hæmorrhage beneath the pleura; but on cutting across it we find that its nucleus is of a rusty red colour, and in the very centre we see signs of commencing suppuration. We may find another area in which the suppuration is more advanced, and yet another in which the centre has softened down to form a distinct abscess. In cases of pyæmia of long duration we find small cavities containing pus surrounded by a thin lining membrane, limiting the extent of suppuration and so showing a tendency to cure.

Pyæmic abscesses are by far most commonly met with in the lungs—in fact, the lungs rarely escape; but we find them also in the brain, the liver, and the spleen; and in some cases we may find them in various parts of the body. We may find the brain riddled with them, many abscesses in the lungs, a small abscess in the wall of the heart, which has given rise to suppurative pericarditis; a small abscess in the wall of the intestine, which has set up suppurative peritonitis; a pyæmic suppuration just beneath the pleura, which has set up empyema; and the case has been admitted with the idea that it was primarily one of empyema; the suppuration may involve the eye, it may involve the skin, it may occur in any rart of the body.

LECTURE XVII.

PURPURA.

Purpura is a condition in which we have to deal with death in the blood. It has been to me an astounding experience to find in the post-mortem room the body of a person, young in years, fairly nourished, and the only visible evidence of disease was here and there a small hæmorrhage. We see cases in which people lose pints of blood and recover—sickly, weakly people go on losing blood for weeks at a time, and keep about all the time and recover—and we know that in cases of purpura there must be something more than the mere loss of blood to kill.

Why does the blood pass out of the vessels in purpura? In one case I examined the capillaries very carefully, and I found evidence of fatty degeneration in the walls of the vessels. I showed the preparation to a friend, a man of large experience, and he said that the changes might be only a consequence of the disease, not a cause. I felt that I must admit the truth of his view, for in no other way could I account for the fatty degeneration. Whenever there is great cachexia or anæmia we find fatty degeneration in the walls of the vessels. Well, some have found in a few cases of purpura evidence of lardaceous disease. "Oh," they said, "here is the cause of purpura—lardaceous disease of the vessels." But they soon found that in most cases of purpura there was no lardaceous disease, and they had to give that theory up. We do not know the cause of purpura.

Now I will give you an illustrative case of purpura. A young woman was admitted into the hospital for pelvic hæmatocele, and was recovering, when she got hæmorrhage into the eye. She again seemed to be improving somewhat, when she got hæmorrhage into the skin; then came hæmorrhage into the brain, and she got paler and paler until she died. The post-mortem examination showed no cause of death but purpura. On inquiry, I found that she belonged to a family of bleeders.

There are some families known as bleeders, for they are liable to death by hæmorrhage from the most trifling wound. Usually in the members of such families we find that there are signs of the hæmorrhagic tendency apart from injury, the males being liable to epistaxis, the females to menorrhagia. Now the history of such people shows that they are extremely and peculiarly sensitive; they are liable to death by mental shock, they are liable to extreme strains of the nervous system, killing them by pneumonia; they are liable to purpuric conditions, and they have the hæmorrhagic tendency in more or less degree. I was led to these views many years ago when I was studying epistaxis very carefully; I used to ask nearly every patient about epistaxis. Very often such a constitutional state is associated with a tendency to phthisis, and therefore we find that epistaxis and a tendency to phthisis commonly go together. In others there is a tendency to rheumatic fever. I remember a young man with rheumatic fever, and he had profuse epistaxis, and the fever suddenly disappeared. These purpuric patients are very sensitive people, and you will often find that before the onset of the purpura they have been exposed to nervous strain. I remember the case of a man in fairly comfortable circumstances at first, but he wished to live above them; he must wear a black coat, he must get into debt, and no one must know about it; he gets more and more involved, and dies of purpura. The antecedent nervous exhaustion seems to affect the vessels and to cause the blood to perish. But in other cases the antecedent condition is more difficult to trace.

Purpura may be ushered in with aching pains in the limbs and swelling of one or more joints, and it is thought to be rheumatic fever coming on, but in about forty-eight hours the gums begin to bleed, and if arrested in one part the bleeding begins in another; it is uncontrollable, but the gums appear normal. Then there are hæmorrhages in the skin, then blood is passed in the water, then blood is coughed up; this is repeated day by day, but the temperature remains normal; we try ice, we try ergot, we try gallic acid, we try one thing after another, but nothing arrests the hæmorrhage.

In the course perhaps of two or three weeks the patient dies, and, as I said before, we are astonished to find on the post-mortem table a body well nourished and not showing any sign of organic disease. We find extravasated blood-clots in the brain, the lungs, the kidneys, and in other organs, and we say that death is due to purpura.

In a case of that kind we may find hæmorrhages into the joints, but it is most important to remember that in death in the early stage of pyæmia we may find similar hæmorrhages into the joints. If death occurs in a few days from pyæmia, at the very outset of the arthritis, we may find hæmorrhages into the joints; the patient having died before there was time for suppuration to occur.

Such cases as I have described to you are usually spoken of as idiopathic purpura, but there is another kind of purpura. Purpura also occurs under conditions which manifest beyond all question that the body was antecedently dying. Purpura sometimes comes on in cases of cirrhosis of the liver; dropsy has been present for some time, and the patient has been getting weaker and weaker, and then purpura comes on and kills. Purpura may similarly lead to a fatal termination in cases of Bright's disease, and we see it also at the end of phthisis. It may occur in extremely exhausted people in connection with rheumatic fever, and you will not forget that we sometimes see it in small-pox, and in malignant scarlet fever; it is also seen in connection with tertiary syphilis.

You will be asked how you would distinguish purpura from scurvy. This is most important in relation to prognosis and treatment. If it is a case of scurvy we shall order lime-juice and fresh vegetables, and most probably the patient will recover. The distinction between them has been worked out both in the wards and in the post-mortem room. In scurvy, sloughing comes in on the gums; there is not merely, as in purpura, bleeding from a pale-looking surface, but the tissues of the gums become charged with blood-clot, purplish-brown in colour, followed by sloughing. A woman was admitted into the hospital with hæmorrhage into the cheek on one side and bleeding from the gums. We could not at first be positive as to the nature of the case; we thought it might be purpura, but in about twenty-four hours I noticed that the centre of the patch in the cheek had become of a brownish-grey appearance, and I knew at once that it was scurvy. A similar patch formed in her throat, then she began to pass bloody motions, and at the post-mortem examination I found the black ulcers of scurvy in the intestine. Numerous hæmorrhages had occurred in the large intestine, and these had sloughed. There is a drawing of the intestine in our museum.

LECTURE XVIII.

HODGKIN'S DISEASE.

HODGKIN'S disease, or lymphadenoma, is a disease of the blood-making organs and great lymphatic structures. The lymphatic glands and the spleen are the common seats of the disease, but it may attack the thymus gland, and spread thence to the pericardium; it may attack the lymphatic system of the kidneys, and produce a characteristic form of kidney disease; in some cases it is remarkably localised, as in a case which was considered to be an outgrowth from the ilium, but after death it was clearly shown that the tumour was lymphadenomatous in nature. At the outset lymphadenoma may be mainly a glandular affection, and in succession the liver, the spleen, and the kidneys, may become affected. In another case the glands may at the outset be comparatively little affected; the patient is admitted to hospital for weakness and shortness of breath, and there is much increased pericardial dulness, and it is considered that the case is one of chronic effusion into the pericardium, and probably due to tubercle; weeks go by, and the area of dulness over the front of the chest increases, but it is noticed that the glands above the clavicles at the root of the neck have become enlarged, and that the spleen has become enlarged, and then we know at once the true nature of the case—that a lymphadenomatous growth has begun in or about the thymus, and spread into the wall of the pericardium, enormously thickening it. Lymphadenoma may be manifested by grey bodies about the size of pins' heads scattered through the lungs. We cannot distinguish the appearance of the lungs from that produced by miliary tubercle, but we find characteristic lymphadenomatous growth in the spleen and other parts. A patient was in this hospital, under the care of one of my colleagues, with firm brawny masses in the skin of many parts of the body, and they extended so that the skin became enormously involved; on cutting into the affected areas a milky-looking exudation followed; it was lymphadenoma of the skin, and the patient died from it. I have no doubt that it had begun in the subcutaneous areolar tissue. Lymphadenoma may begin as a localised growth with very little sign of antecedent cachexia; on the other hand, it may begin with very little evidence of localised growth, but with extremely marked cachexia.

In the cases that are predominantly characterised by glandular enlargement, usually the first symptom that is noticed is swelling of some gland. It may begin in any of the lymphatic glands; it may be in the glands of the neck, in those of the groin, in the thoracic, or in the abdominal glands; and in these cases the first question we usually have to ask ourselves is, "Is this enlargement of the glands strumous, or is it the beginning of another morbid condition that will sooner or later prove fatal?" Strumous glands go on enlarging for months, and then subside, and the patients recover; and this sequence may be repeated again and again over years; we do not therefore attach much importance to this condition. Lymphadenoma has a very different history, so that we have to keep our minds very clear about the characteristic signs of lymphadenoma in the glands, lest we should mistake it for struma.

We are impressed at the outset by the hardness of the glands. We do not feel an elastic enlargement, as when there is recent strumous swelling, but there is a firm, hard, stony condition, as if there were much fibrous growth. We must bear this in mind, that if a strumous gland has been a long time enlarged it may be converted to a great extent into fibroid tissue; so we ask the patient this question, "Has this enlargement of the gland existed for many months or years?" If the enlargement is of long duration it is opposed to the supposition of lymphadenoma, and the case is more probably one of strumous enlargement with fibroid thickening. We may think it wiser to give no decided opinion for a while, but we notice that gland after gland is becoming similarly affected, similarly enlarged and indurated, until, if it is in the neck, there is a continuous chain of enlarged glands; and we notice another feature that is still more suggestive of lymphadenoma, that the glands are becoming immovable, that they are fixed together, and fixed into the surrounding tissues.

Hodgkin in his original description pointed out that the growth extended through the capsule of the gland into the surrounding tissues, thus giving rise to the fixed condition of the glands, and this, he said, being like cancer, led him to regard this glandular affection as a malignant disease. Like a malignant disease, the growth is steadily progressive until it kills.

As the glands increase in size, and more and more of them become involved, we observe that the patient becomes paler and paler. In Hodgkin's disease there is usually much anæmia, and especially in these glandular cases, and for this reason the disease was years ago called lymphatic anæmia. As the disease advances we may observe that the glands of the axilla are becoming enlarged and indurated, and that the glands of the groin also become involved; there is increasing weakness and increasing shortness of breath, and the question arises, "Is the shortness of breath merely due to the anæmia, to the loss of red corpuscles, the great breathing organs of the blood, or are there also lymphatic growths within the chest?"

Months may thus pass over, the patient by rest gaining strength to some degree, with consequent relief to the breathing; but there is evidence of increasing growth in the various lymphatic glands of the body. There comes a time when we find that the liver and the spleen are felt below the ribs, and the spleen goes on increasing in size, so that we ask, "Is it leucocythæmia?" We examine the blood, and we find that there is no great excess of white corpuscles. As the spleen enlarges we may observe that its surface becomes uneven, and the same condition is noticed in the liver; we feel masses of induration, so that we know that there is growth going on within the substance of these glands. We may next observe that there is albumen in the urine, leading us to think it probable that the lymphadenoma is affecting the kidneys.

The case advances with increasing anæmia, until the weakness and shortness of breath are so extreme that the patient can no longer leave his bed.

What do we find on making a post-mortem examination? The colour of the glands resembles that of parsnip, and they cut in the same way, but are somewhat firmer; when squeezed, a little milky juice flows out, but there is very little; it is not a very juicy growth, like cancer, the whole gland seems to be converted into this greyish-yellow fibroid material. We notice that the capsule of the gland is similarly affected, and that the growth has invaded the surrounding cellular tissue and the muscles. There is little or no caseous matter—I think I may say there is none at all; and this is one of the great distinguishing features from struma. In strumous glands we find masses of caseous matter, but none in lymphadenomatous glands. Let me repeat, that, if there has been long-continued strumous disease of glands, we may find, in addition to the caseous matter, much fibroid substance, burying the caseous material, and binding the glands together in a hard firm mass extending down the neck, so that during life the appearance strongly resembles that of lymphadenoma. We had a boy in George Ward in that condition, and for months we could not determine whether it was lymphadenoma or not, but when he died we found that there were caseous remnants buried in the fibroid tissue. I may here mention that in struma we may find caseous tubercular masses in the liver and in the kidney; similar anatomical structures are affected in struma and in lymphadenoma.

In some cases of Hodgkin's disease in which the morbid growth has been more rapid, the glands are softer; they seem to be made up of a white pulpy material, with here and there much congestion, and even hæmorrhage; but in such a case, in addition to the softer glands, we usually find some glands in the firm, hard condition I have described.

The bronchial glands are commonly affected in Hodgkin's disease, and when enlarged they more or less obstruct the bronchial tubes; the retro-peritoneal glands and the mesenteric glands may also be affected; in fact, the glandular system throughout the body is liable to suffer. When we cut into the spleen we observe circumscribed greyish-yellow masses of a similar firm fibrous substance scattered here and there, ranging in size from that of tubercle up to an inch or more in diameter. In the liver we find similar masses. The kidney has a very characteristic appearance. You will find in the New Sydenham Society's Atlas of Pathology a drawing of a lymphadenomatous kidney, taken from our post-mortem room. The kidneys are usually very large, and of a mottled appearance. There is much congestion on the surface. When we cut into the kidney we find that the demarcation between the cortex and the cones is obscured, owing to the extensive invasion of the kidney substance by the lymphatic growth. The kidney seems to be converted almost entirely into a morbid lymphatic growth.

What do we find on examining this material by the microscope? We see enormous numbers of lymph-cells, but instead of lying in the meshes of a very finely fibrillated matrix (adenoid tissue), we find that there is a great excess of fibroid tissue amongst the cells; there seems to be an increase of the lymphatic structure, but with a great preponderance of the fibrous element. It has been familiarly spoken of as an hypertrophy of the glands, and in a sense it is, for the normal structures are repeated in excessive quantity; but there is this great distinction from the normal tissue, that the fibrous elements of the gland are much increased, and hence the new growth is very tough. This is the very reverse of what occurs in struma. In struma there is an enormous increase of cellular elements, and they get so tightly packed that the lymph can no longer circulate freely among the cells, and they become imperfectly nourished, and undergo fatty degeneration, so that the gland is changed into caseous matter.

In these cases of lymphadenoma, as in ordinary fatal cases of anæmia, death is usually due to fatty degeneration of the heart.

I will describe another kind of case. The disease is the same, but takes a different anatomical course. The patient becomes more and more anæmic, and the doctor is at a loss to account for the persistent and increasing anæmia. From time to time there is a rise in temperature; the temperature may remain for a few days at 101° or 102°, and then fall to normal again. He may notice at the same time that the spleen is somewhat enlarged; shortness of breath becomes troublesome, and on examining the front of the chest he finds dulness, more particularly marked over the sternum, and extending towards the apex of the heart—in fact, over the cardiac area. He notices also that the heart sounds are getting more and more muffled, they have a distant character, and he wonders if there is fluid in the pericardium. Finally, the heart sounds may become entirely inaudable. From time to time there is a recurrence of the feverish outbursts, lasting one, two, or three weeks, and there is progressive anæmia.

After a time he may notice that one or two of the glands about the clavicle are enlarging, and then at once he begins to suspect the true nature of the case, that it is one of lymphadenomatous growth of the pericardium. Later there is enlargement of the liver and spleen, and the nature of the case is beyond doubt.

What do we find on making a post-mortem examination in such a case? When we raise the sternum, a greyish-yellow firm mass is seen lying in the position of the thymus gland, and this has extended into the pericardium, so that the anterior wall of the pericardium has been converted into an immense fibroid mass, it may be as much as an inch thick, and the thickest part is over the base of the heart, in the region of the thymus gland. As I have already told you, the growth is rather dry, not very juicy, like a cancerous growth. We further notice that the morbid growth has extended from the pericardium into the anterior margins of the lungs, and then we say at once, "That is like a case of Hodgkin's disease, as it extends from one structure into the surrounding ones." On cutting into the lungs we find them studded with bodies that resemble miliary tubercles, but in addition to these small deposits there may be one or more of a much larger size. The liver and spleen are affected as I have already described.

In a third group of cases, a tumour forms in some part of the body; it is firmly attached to the surrounding structures; it does not grow very rapidly; it is not so nodular as an ordinary carcinomatous growth; it grows slowly, is exceedingly firm, and is evidently malignant from the way in which it spreads into surrounding structures. No remedies arrest it; there is increasing anæmia; after a time the lymphatic glands in various parts of the body become enlarged, and the spleen begins to swell, and we decide that the case is most probably one of lymphadenoma. Usually in these cases the diagnosis lies between struma, lymphadenoma, and medullary cancer. In some cases medullary cancer is very widely spread in various organs; there is tumour growth in many lymphatic glands, in bone, in the spleen, in the liver, in the lungs, with feverish outbursts and increasing anæmia. What usually helps us to a diagnosis in such cases is that the cancer is a much more rapid growth; there is a subacute formation of cancer; the tumours are not so hard, being much more vascular, so that there are sometimes subcutaneous hæmorrhages from the vascular growths. I have already dwelt upon the distinguishing characters of struma, and need not return to them.

If you ask me the causes of lymphadenoma, I can only tell you that the causes are unknown. The disease occurs more commonly, I think, in young people than in middle-aged, and I have never known it occur in very old people. There has been some confusion between Hodgkin's disease, or lymphadenoma, and leucocythæmia. This latter, however, is quite a different morbid condition, characterised by an enormous excess of colourless corpuscles in the blood, leucocythæmia, white-cell blood, or leukæmia, white blood. The confusion between Hodgkin's disease and leucocythæmia arose because some observers said that in the former disease, as well as in the latter, they had found an excess of leucocytes in the blood; but if there is an excess, it is nothing very striking. In ordinary anæmia, and in struma, as well as in Hodgkin's disease, we may find an excess of colourless corpuscles in the blood, but this is very different from the state of the blood in leucocythæmia. In this disease the blood at first sight seems to contain only white corpuscles; it is not a case of one white to a hundred red, but the number of white corpuscles is enormously increased. I have never seen anything like that in Hodgkin's disease. I shall tell you more about leucocythæmia when I come to lecture on diseases of the spleen.

LECTURE XIX.

ADDISON'S DISEASE.

ADDISON'S disease is characterised by a grey, firm morbid growth in the supra-renal capsules. Before discussing the question which many years ago excited so much interest, whether the changes in the capsules are the main element in the causation of Addison's disease, a question which divided the medical profession into two camps—the physicians of Guy's Hospital maintaining with Addison that this question must be answered in the affirmative, and the physicians of University College Hospital answering it in the negative—it will be well to describe the morbid appearances of the capsules.

If the patient dies in the early period of the disease we find the capsules much enlarged. Notice that I use the plural; if one capsule only is diseased, it is not a case of Addison's disease. The capsules are thicker, heavier, and tougher than normal; the enlargement and toughness of both the capsules immediately lead us to think of Addison's disease. On cutting into one of the capsules we see that the pigment layers have almost disappeared, whilst the grey middle layer is much increased in size. The organ is infiltrated with a grey material which was formerly called lardaceous, and it does look like lard, but is of much firmer consistence, and it is not lardaceous in the sense in which we now use the term. The mass consists of elongated spindle cells, the growth of which has begun in the middle layer and extended into the pigment layer. With such morbid appearances we know that death has taken place at a very early stage of the disease, and we are not surprised to find little or no abnormal pigmentation of the skin.

If the course of the disease has been very chronic, so that the patient has lived for years, parts of the fibrous substance undergo caseous degeneration and other parts contract, so that we find that the capsules are less enlarged; there is the same grey substance, but less of it, and scattered through the capsule are caseous areas. In some cases we may even find calcareous masses. In the most protracted cases of Addison's disease the contraction of the capsules is extreme, so that I have actually had some difficulty in finding their remains.

Now let us, before going any farther, ask if there are any characteristic symptoms associated with these changes in the supra-renal capsules, and if so, such symptoms are found in no other connection. First, let us consider other destructive morbid processes affecting the capsules, and describe the symptoms, if any, associated with these. On post-mortem examination we not uncommonly find extreme venous congestion of the capsules, and consequent hæmorrhage into their substance. This condition produces no known symptoms. Tubercular disease of the capsules is common. Both the miliary tubercle and the yellow caseous tubercle are found in connection with phthisis or with general tuberculosis. No special symptoms are associated with these changes. Lardaceous disease also occurs. This invariably affects both capsules. We find the capsules infiltrated with a suetty-looking material, and they are firmer and heavier than normal. This change is found when there is wide-spread lardaceous disease, in which the liver, the kidneys, and the spleen are involved. It is therefore incidental to lardaceous disease of other organs, and produces no special symptoms. A characteristic gummous formation may be found in one or both capsules in cases of tertiary syphilis, but we learn that there have been no characteristic symptoms pointing to disease of these organs. Cancer of the supra-renal capsules also occurs, and, like the last mentioned, has no definite symptoms.

We return then to ask if the symptoms found in connection with the morbid changes in the capsules of the nature first described are definite and characteristic. This question must be answered in the affirmative.

What is the suffering in such cases? There is always one leading symptom—extreme failure of circulation. This is a marked feature alike in the subacute and in the chronic forms of the disease. We can find no evidence of any organic disease, but the patient from time to time is nearly pulseless, and he often feels so weak and so giddy, that he can stand upright with difficulty, and in extreme cases can hardly raise himself in bed. The medical man says to himself: "I cannot account for this miserably small pulse; there is no evidence of gross organic disease, and yet at times the pulse becomes so soft and so small that I have to exercise great care in counting it." That is the common history, and such a history at once leads us to think of Addison's disease.

With this weakness in the general circulation there is usually much nervous restlessness. There may be vomiting from time to time, or an occasional attack of diarrhœa. Apart from these transient disturbances, the patient takes food much as usual, the bowel acts much as usual, the temperature remains normal, and there is no great loss of flesh, unless the case is of very long duration. In the attacks of extreme prostration there is a clear and seemingly calm mental condition, for these patients do not complain much. In many cases the suffering I have described passes away after two or three weeks, and such attacks may recur for months or even years, and in each attack the prostration is so extreme that we always fear that it will pass into coma.

As collapse comes on, restlessness is very marked; as the circulation in the brain is failing the patient tosses about, and is unable to lie still until coma comes on. One Sunday morning I was going round the wards at Guy's Hospital with Dr. Wilks, who had made a special study of Addison's disease under Addison himself. Dr. Wilks pointed to a man moving restlessly about in bed, saying, "I do not know what is the matter with that man, but from his restless appearance I cannot help thinking that he is dying from Addison's disease." The man died next morning and it was a case of Addison's disease.

At the post-mortem examination in such cases we find the characteristic changes I have already described. The left ventricle is firmly contracted and empty, as in death by collapse, showing us that the blood has been cut off from the left ventricle and from the arteries. There may be pneumonia, and in long-standing cases we may find chronic caseous destructive changes in the lungs, but there does not seem to be any special connection of these changes with the disease, for we find such changes in death from many causes. In one case I found acute pericarditis, but we must remember that there is a liability to the onset of acute inflammations in many forms of dying.

Now I come to the answer of the second part of the question I asked above, whether the symptoms of Addison's disease are always connected with disease of the supra-renal capsules. We have seen that there is a very definite group of symptoms to which we give the name of Addison's disease; we have seen that cancerous, tubercular and syphilitic destruction of the supra-renal capsules does not lead to the onset of these symptoms; we have described the appearances of the capsules met with in many of the cases of Addison's disease. But, we have to ask, since destruction of both capsules does not necessarily give rise to the onset of Addison's disease, must there not be another element than disease of the capsules in the production of Addison's disease? Now it has been clearly recognised that after death from Addison's disease we may in some cases find little or no change in the supra-renal bodies. In some of the most rapid cases of Addison's disease we find least change in the capsules. This led to the search for evidence of morbid changes around the capsules, and on dissecting out the nervous communications between the capsules and the semilunar ganglia and the great nervous plexus of the sympathetic in front of the aorta, evidence was found of a chronic inflammatory condition of the nerves and ganglia. In some cases there was so much milky fibroid tissue that the remains of the semilunar ganglia were only discoverable with the microscope. Disease of this great organic system of nerves will account for the vomiting and diarrhœa. Further, we know that whenever there is disease within the abdomen leading to pressure on the semilunar ganglia there is a liability to the rapid onset of fatal collapse.

We know also that great irritation in that region, which may perhaps be evidenced only by the patient's suffering, may kill by coma or by other brain failure. Such patients often kill themselves.

We must connect Addison's disease with suffering in the epigastric region. Never underrate the importance of such suffering, which patients will often describe to you by saying that it is indescribable. You will often meet with such suffering in practice, and will have carefully to ask yourselves, how much food, how much rest, and how much warmth do these patients want?

You will have been surprised that I have not spoken of pigmentation of the skin, but I wished to defer a more exhaustive consideration of this symptom to the last. We have been taught that pigmentation is a constant symptom in Addison's disease, and therefore we always look carefully for it; but we may be unable to say that there is anything abnormal about the pigmentation of the skin, and the excessive pigmentation may be manifested only by the mucous membrane of the mouth. In the case I spoke of just now, that I saw in Guy's Hospital, the only part in which there was abnormal pigmentation was the mucous membrane of the mouth. In thinking of pigmentation I am always much impressed by a case which was in this hospital under the care of Mr. Hutchinson, who had specially studied the subject of pigmentation of the skin in Addison's disease, so that you will understand that his mind would be ready to recognise the disease. Now there was a patient of his with bone disease, and vomiting came on, and the circulation rapidly failed, and the patient died. When I made the post-mortem examination I discovered, what had never been suspected during life, that it was a case of Addison's disease. There was some abnormal pigmentation of the hands and wrists, but it was so slight that it had been entirely overlooked.

I will give you two more cases. There was a patient I was asked to see in consultation, a young man who worked in a shop in this road. His medical man said to me: "It is a most obscure case; he was at work only a few days ago, and is desperately ill now, and I cannot account for his miserable pulse." He was well nourished, but the pulse was so small that it was felt with difficulty. There was little or no abnormal pigmentation of the skin. He died very shortly, and I saw the supra-renal capsules myself. There was no doubt that it was a case of Addison's disease.

I saw another patient at Stratford whose skin was just the reverse of this. This man was the son of a baker. He was so weak that he nearly fell down in the street at times, and often felt so giddy that he had to sit down. His skin was deeply bronzed all over. I remember at one time, when my experience of Addison's disease had only been of cases with very little pigmentation, I saw a print in an atlas of a case with the skin so deeply bronzed that I was inclined to doubt if the case was really one of Addison's disease, but I asked Dr. Wilks about it, and he told me that the drawing was taken from a woman he had known quite well, and that it certainly was a case of Addison's disease. Now the baker's man was like the case in the drawing, there was extreme bronzed pigmentation all over the skin.

Now what were the other points of difference between these two cases? In the case in which there was little pigmentation the disease had run a subacute course, and at the post-mortem examination the capsules were found to be large and firm, the change consisting in a fibro-cellular thickening of the middle layer of the capsule. The baker's man had been under the care of Mr. Nicholson, of Stratford, for some years, and after death the capsules were found calcareous and contracted. The conclusion I drew from these cases was that the degree of discoloration depended on the duration of the case. To a certain extent these conclusions were justified, but I think it is very difficult to connect the different degrees of pigmentation with the degree of morbid change in the supra-renal capsules, and the chief thing I wish you to bear in mind is that the pigmentation is extremely variable.

A man was admitted into the wards with pigmentation very similar to that of Addison's disease as far as colour was concerned. There were patches of a very dark tint scattered over the skin. The skin where not pigmented was notably pale. His pulse was small, so that we could only count it with difficulty. He had diarrhœa like that of Addison's disease, and became weaker and weaker; he was extremely restless. I remember dictating a note something like this: "This case is in many ways like Addison's disease, but I do not think that it is that disease, because the pigmentation occurs in patches." Addison himself described the pigmentation as resembling that which is seen when a person has been much exposed to the sun in a tropical climate. It is most distinct in exposed parts, the neck, the face, the hands and the wrists, and it may occur in an extreme degree all over the body, but it never occurs in circumscribed patches. Now in the case of which I was speaking the pigment was distributed in patches. The patient died from fatty degeneration of the heart, and that was the only disease I found in the body. I examined the capsules, and the only thing abnormal was that one of them contained a body about the size of a small shot, which seemed to be a little tumour, and this was only in one capsule, whereas in Addison's disease the morbid change always affects both capsules.

Remember then that in Addison's disease, in parts normally pigmented the pigmentation becomes extremely marked, and in the early stages we look out for excessive pigmentation of the areolæ of the nipples, the anterior folds of the armpits, &c. There is a similar pigmentation of the mucous membrane of the mouth, and therefore, if from the condition of the pulse you suspect Addison's disease, never fail to examine the mucous membrane of the mouth. But in thinking of Addison's disease do not forget how extremely variable is the pigmentation.

Now let us go on to ask what clinical experience teaches us with regard to great pigmentation of the skin. Is it a condition to which we can attach any great practical significance? We find that pigmentation of the skin occurs in an extreme form in various diseases characterised by great asthenia. Look out for cases of malarial poisoning as you go through the wards, and you will find some such patients with the skin extremely dark brown, almost black, with a pulse hardly to be felt, the voice so feeble that it is reduced to a whisper, the temperature below normal, and the whole appearance strikingly resembling that of Addison's disease. Nurse such a patient carefully, keep him in bed and give him rest, and his circulation will probably be re-established, and the dark colour will clear away, so that in a week or two there will be a wonderful difference in his appearance. This condition is well recognised to occur in connection with severe malarial poisoning. In the less severe and more chronic cases of malarial poisoning, known as malarial cachexia, we find pigmentation of the skin associated with great asthenia, there is a small pulse, and a tendency to attacks of vomiting, just as in Addison's disease. We are led to think that in these malarious conditions there is a great blood change, a peculiar death in the blood, perhaps due to the presence of some poison.

Now I will give you another kind of case—pigmentation of the skin in connection with morbus Brightii. I had under my care a patient with this disease who had a skin quite as dark as any I have seen in Addison's disease, and as the general circulation got stronger and the œdema disappeared, the pigmentation of his skin passed away. Granular contracted kidney and pigmentation of the skin are frequently associated.

Some of the most extreme forms of pigmentation of the skin I have ever seen have been in cases of phthisis with great asthenia; usually these were severe cases of phthisis tending rapidly to death. Pigmentation of the skin is often met with in the cancerous cachexia. We meet with it sometimes in connection with chlorosis. Lastly, I must mention the pigmentation of the skin that occurs in nervous diseases, and which is very marked in some cases of hysteria. In many such cases you will notice pigmentary deposit in the skin beneath the eyes, and we may estimate the degree of disturbance of the mental condition by the degree of pigmentation of this part. We notice pigmentation also in other nervous diseases, notably in various forms of insanity.

We are then led to see that pigmentation of the skin is associated with various morbid conditions, in which there is marked failure of the circulation of the blood or of the lymph, or marked failure of the nervous system. The skin being so intimately connected with the breathing organs, its nervous structures promoting respiration, whenever there is great failure of breathing, as in phthisis, we are not surprised to find pigmentation of the skin.

There is another morbid condition of which I should speak in connection with pigmentation of the skin, and that is, heart disease. In some cases of dilated left ventricle we notice a yellow colour of the skin. It looks like jaundice, but we cannot detect bile in the urine, and after death we cannot find any sign of hepatic disease to account for jaundice. We see a similar coloration of the skin in some cases of pyæmia. I think that the appearance of this colour means death in blood, and therefore it is a most serious sign.

LECTURE XX.

SYPHILIS.

The first thing that we notice after the primary infection is that the glands in the immediate vicinity of the infected part become swelled, tender, and elastic, and in some degree painful. We sometimes fear that they may suppurate, but suppuration of the glands in the groin belongs rather to gonorrhœa than to syphilis. After the glands nearest the infected part have become inflamed, more or less swelling of the glands in other parts of the body follows. In some cases permanent thickening of the glands is left, so that an indurated gland in the neck may tell of the previous existence of a syphilitic throat, or from the existence of such a gland in the groin we may infer that the patient has had a chancre on the penis.

During the primary infection the local induration may be so little marked, that it may, even in the penis, be entirely overlooked, so that it is generally recognised that even a reliable man may be mistaken in saying that he has never had a primary syphilitic sore. After the primary stage there is an interval of comparative freedom from suffering. I must not say that the patient is healthy, but there is no recognisable disease, and then after some weeks—let us speak cautiously, for opinions vary—the secondary symptoms appear. Congestive conditions come on. We see congestive patches on the skin, the usual coppery mottled eruption. The congestion may lead to more thickening of the epidermis, so that we get conditions resembling psoriasis, &c. There is a similar congestive condition of the throat, with thickening of the mucous membrane, and white, or rather grey, patches of superficial exudation. There may be a similar congestive condition of the windpipe, giving rise to local symptoms, or there may be congestion of the bronchial tubes, with symptoms of bronchitis. There may be a similar congestive condition of the kidneys. After a time, varying according to the general health of the patient, these symptoms pass away. If there is much cachexia the secondary stage is usually much protracted, but finally the symptoms entirely disappear, and the patient is free from objective signs of disease; I must speak very cautiously here, being on very debatable ground.

From this time months or years may elapse before the tertiary symptoms appear. If the patient becomes debilitated from any cause the tertiary symptoms are apt to come on, and a curious feature is that the tertiary disturbance may be very localised. It may be limited to a small patch of skin, or to the windpipe, or to one of the testicles, the other organs being comparatively little or not at all affected. It may attack a small vessel of the brain, leaving by far the greater part of the brain free from any morbid manifestation. Again, it may be that the change is localised in the sheath of a single nerve, and I might multiply instances.

Tertiary syphilitic changes are, on the post-mortem table, most commonly witnessed in the liver. You may be asked to describe the morbid appearances of syphilitic disease of the liver. Let me first mention that in infants there may be a subacute syphilitic change leading to swelling of the liver. We find a large homogeneous-looking liver, with its colour more or less altered, and on microscopic examination we see an enormous number of cells, with large nuclei, and containing many fat granules, scattered throughout the liver substance, which have evidently originated from the connective tissue of the liver. In such cases the infant usually dies with jaundice, but it does not always prove fatal. In adults who have had syphilis, tenderness may come on in the region of the liver, no acute pain, but some aching; the liver-dulness may be somewhat increased, and we think that there is a congested subacute inflammatory condition of the liver, and we wonder as to the cause. We do not find any history of great abuse of alcohol, there is no reason to suspect hydatid, nor have we any reason to think it is malarious, we are then led at once to think that it is syphilitic. After a time the symptoms may pass away. There comes a time when the patient has a permanently enlarged liver; on feeling the surface through the abdominal wall we are not quite sure that it is not a little altered, but it is for the most part smooth, there is certainly no extreme nodular condition. The liver extends several inches below the ribs, and is somewhat harder than normal, and we find nodes or other evidence of tertiary syphilis in other parts of the body. There is another group of symptoms, characterised by signs of contraction of the liver, the liver dulness is diminished. If the patient takes a very deep breath it may be possible to feel the edge of the liver below the ribs, and if so we are struck by its indurated and nodular feel. We notice that the belly is increasing in size; ascites comes on. You see that there are two distinct groups of cases, those with a large liver and no ascites, in such a liver there may be contracted patches here and there, but they are very limited in area, whereas in the other cases there is an extremely contracted liver with ascites. What are the post-mortem appearances? In the smallest and most contracted and diseased syphilitic livers we find the least gummous change. These livers have large deep puckerings on their surfaces, and between the furrows there are portions of the liver projecting in the form of large nodules. It is notorious that these nodular masses may be mistaken clinically for cancer. We feel through the abdominal wall that the liver is nodular, there is a good deal of wasting and ascites, so that we may easily set the case down as one of cancer. These patients may recover, the dropsy disappears, and they may live for years, so that it is a mistake to give an unfavourable opinion. On cutting into the puckered part we notice that the capsule of the liver is thickened by a grey fibroid substance, and that the fibroid tissue spreads widely through the liver substance, forming an irregular network. This fibroid tissue is most abundant where the natural connective tissue of the liver is most present, and we therefore find less of it in the centre of the liver than close under the capsule. On examining this fibroid tissue closely we may find here and there small deposits of wash-leathery looking gummous material, but I have several times failed to find any of it. On microscopic examination we find the connective substance of the liver enormously thickened by fibroid material. You might on post-mortem examination mistake such a liver for the product of cirrhosis, but you may distinguish it by the large size of the nodules, by the extreme puckering, and by the bands of fibroid thickening radiating from the puckered centres. All these puckered centres are small nodes, but are distinguished from the commoner varieties of node by the small quantity of gummous material they contain. In the cases with a large liver and no ascites we find the liver for the most part smooth, but here and there a little superficial puckering, enough to reveal that it is syphilitic; the capsule is thickened, and the liver feels a little firmer than normal, but is not extremely indurated. We are chiefly impressed by its large size. It does not feel waxy, like a lardaceous liver. On cutting across it we observe large masses of yellow gummous material, one, two, or more inches in diameter; the masses are surrounded by some fibroid tissue, but there is no widely spread fibrosis as in the contracted liver; there may be several of these large masses, the growth of which has evidently caused the increase in size of the liver. I cannot say that I have never seen dropsy in such cases, but where I have seen it, it has not been due to fibroid changes in the liver substance, but to the growth of an enormous gummous mass which has pressed on the portal vein. You will gather from what I have told you about these livers that even in the most extreme conditions the syphilitic growth is arranged in nodular masses, leaving portions of liver comparatively free, so that if such portions can get enough blood, we may hope that they will be able to carry on the liver functions sufficiently well to maintain life, and clinical experience bears out this view. There was a man in hospital who had a syphilitic liver such as I have described to you, he was tapped thirteen times, and an enormous quantity of serum was drawn off. I went on hoping. I thought that I must try to get his general circulation stronger, must not discourage him, but rest and soothe him, and get him out into the open air in the intervals between the tapping, not let the fluid accumulate so enormously as to stop his breathing, and see what time would do for him. In the end we discharged him quite comfortable, and able to go back to his work, and as far as I know he is going on well now. The recoveries for a while from tertiary syphilis are wonderful, no matter what part of the body is affected.

How does tertiary syphilis affect the lung? The lung is more commonly the seat of tertiary syphilis than we were at one time disposed to think. The lung affection is very similar to that of the liver, with the exception that I have never seen large gummous masses in the lungs. There is fibroid thickening of the pleura, patchy, like that of the capsule of the liver, the fibroid substance extending into the substance of the lung, and leading to its contraction. Usually the contraction is not extreme, it may contract the lower lobe to a considerable extent, but it rarely leads to very extreme cirrhosis of the lung. I have, however, seen a lung almost entirely consolidated by syphilitic fibroid thickening, and when we cut into it we found only a gristly, firm, tough material. In that case there had been a mass in the breast, and the surgeon had been in doubt whether it was cancer. On post-mortem examination we found that the mass in the breast was a syphilitic gummous tumour, and there was a continuous fibroid mass extending from this through the chest wall into the lung. In rare cases there may be syphilitic gummous masses scattered through the lung in a tubercle-like form, masses like small peas. Most commonly we find the scattered fibroid patches in the pleura and lung, with signs of red and grey hepatisation, the remains of acute pneumonia which has killed the patient. Death by pneumonia is the great risk when there is syphilitic damage of the lung. There is a peculiar greenish-yellow appearance of the hepatised areas which always makes me think of syphilitic pneumonia directly I see it. In rare instances the gummous mass may be much limited, involving a bronchial tube, and may lead to contraction of the tube, and death by broncho-pneumonia. There may be a gummous mass in the trachea, leading to symptoms closely resembling those of aneurysm, and intra-thoracic cancer. There are undoubted signs of constriction of the trachea, no signs of aneurysm or of tumour can be discovered, the signs of constriction become extreme, and bronchitis comes on, which commonly kills the patient, owing to the accumulating exudation and increasing dyspnœa. When we cut into the trachea we find its passage very much narrowed, there is a tough fibroid constricting ring, and in this we find some gummous material, so that we know that it is syphilitic.

Tertiary syphilis attacks the kidney; we may find the fibroid material either in scattered masses, or it may be so widely diffused throughout the organ that the appearance resembles that of granular contraction. You may be asked very pointedly about that, and as it is an observation which is not, I think, very commonly made, I tell you that I consider the fact beyond all question. There was a man whom Mr. Hutchinson had had under observation for years with syphilitic symptoms. I saw him lying in bed, semi-comatose, and was told that he had had albuminuria and other marked symptoms of renal disease, and that uræmia had come on. He died, and at the post-mortem examination I found the kidneys tough, granular, and much contracted. Nevertheless, though there was no doubt that he had died from uræmia, and the kidneys were extremely contracted, the appearance was not that of ordinary granular contraction of the kidneys, the kidney was more fibroid looking, there was the same kind of fibroid thickening that we see in syphilitic disease of the liver and of other parts, and in addition there was this interesting fact, that there was no dilatation of the heart such as we should have found in an ordinary case of chronic Bright's disease.

In other cases the kidney affection in tertiary syphilis takes a different form. I have found evidence of tertiary syphilis in other parts of the body, and on examining the kidneys I have found some fibroid tissue scattered here and there, but the kidneys were large and mottled, with a peculiar greenish-yellow colour intermixed with the red, so that I immediately suspected that there had been syphilitic nephritis. It is evident that in the kidney as in the lung acute inflammation may arise in the organ damaged by fibroid thickening and kill the patient. Remember, then, that if there is evidence of tertiary syphilis, and the patient is getting weaker, you should never fail to examine the urine, and you should also carefully examine the lungs, for there may be an insidious pneumonia going on, or a very insidious nephritis.

You will find it easy to remember what I have to tell you about syphilitic disease of the testicle, if you bear in mind what I have told you about the changes in the liver. We may find the testicle extremely tough and contracted, and when we cut into it we find a quantity of grey fibroid material, always most marked at the back of the testis, spreading into it from the epididymis. We may be quite unable to find any gummous material, but the dull white-grey colour of the fibroid tissue, similar to that which we see in syphilitic scars of the skin, at once tells us that it is syphilitic. In other cases we find a quantity of yellow material scattered in the testicle, surrounded by fibroid tissue, and we have to decide whether it is a strumous or a syphilitic testicle. If there does not happen to be evidence of syphilitic disease in other parts of the body, we may be unable to come to a decision, but the yellow gummous material of syphilitic disease is firmer than that of strumous disease, and that is why I speak of it as leathery material. I want you to think of the toughness of wash-leather.

Syphilitic disease of the periosteum is one of the commonest of tertiary changes. The periosteum is thickened by the accumulation of fibroid material, so that its surface is raised. The tibia is often affected, and if we pass the hand along the anterior edge of the bone we feel it is raised into little mounds. The change may extend more or less into the bone, the bony substance becoming condensed by increased ossification. In connection with this let me give you a matter of general experience: any morbid growth in the periosteum may become ossified, so that the appearance of a bony outgrowth is produced. So we often find these swellings of the periosteum ossified in syphilis. We may find gummous material scattered among the fibroid thickening.

The endosteum of the cranial bones, the dura mater as it is called, becomes similarly affected with fibroid and gummous material, and the pia mater and the surface of the brain may also be involved by the thickening, resembling that of the capsule of the liver and of the pleura of the lung. It is this change that is attended by syphilitic dementia. When we saw across the skull we find that the bone is markedly thickened, and the spaces of the diploë filled in by excessive bone-formation

—syphilitic ostitis. On examining the inner table of the skull we are struck by its altered appearance, it looks denser than the normal bone, and is here and there irregularly raised; we are struck by the whiteness of it, it has lost its pink appearance, and has the greyish-white look of syphilitic change. We find widespread thickening of the membranes. Changes in the periosteum of the cranial bones are very common, syphilitic node of the skull. As elsewhere, there is both fibroid thickening and formation of gummous material. Nodes are most common on the tibia and on the cranial bones, but may be found on the clavicle or on a rib—in fact, on any bone of the body.

Syphilis attacks the vessels of the brain and of other parts. When I have at a post-mortem examination found syphilitic changes in the cerebral arteries, the appearances have been such as we could not mistake. There has been a very limited patch of thickening in the adventitious coat of the vessel, making a greyish tumour projecting from the vessel, which has immediately riveted our attention. Its limitation at once led us to suspect that it was syphilitic, the other vessels being normal. On cutting into the mass we found that the thickening extended through the wall of the vessel into the artery, and there was some yellow gummous material amongst the fibroid tissue. The intima had become roughened by the growth, blood had coagulated upon it and plugged the vessel. The portion of the brain supplied by that vessel had softened, and hemiplegia had come on. I have known such a plug spread across to the opposite cerebral artery and lead to a double softening. The cases I have seen have been plugging of the middle cerebral artery, but the disease may affect the basilar or other arteries.

Tertiary syphilis may attack the aorta. You will find a drawing that I made of such a case in one of the volumes of the Pathological Society. We find grey fibroid thickening of the lining membrane of the aorta exceedingly limited, exceedingly patchy, unlike atheroma. The fibroid tissue is much puckered, like that in the liver. If the patches are near the aortic valve, acute endocarditis may come on and kill the patient, or there may be much puckering of the valve, causing incompetence. When Mr. Maunder introduced the practice of tying arteries for the cure of aneurysm and for the arrest of inflammation, it brought to my mind the question, Does syphilitic disease of the arteries lead to aneurysm? It now seems to be an accepted opinion that it does. It is beyond question that we find aneurysm in those who have had tertiary syphilis, and we find that the disease of the artery which has caused the aneurysm is exceedingly limited, like syphilis; and yet I felt it was difficult, even in cases in which there was other evidence of tertiary syphilitic disease in the body, to feel quite certain that the pathological process which had led to the formation of the aneurysm had not been due to other causes than syphilis.

Syphilis may affect the cranial nerves. We find small masses of fibroid syphilitic material on the sheath of the nerve, it may be the third, the sixth, or the seventh, and the compression of the nerve filaments leads to paralysis.

During secondary syphilis, while the eruptive manifestations are going on in the skin, there may be much constitutional disturbance, so much so that we have sometimes wondered if we had to do with a case of typhus fever. There is a mulberry eruption scattered over the body, sordes about the lips, much tremor, a high temperature, and the patient with difficulty able to stand owing to weakness and tremor; but when we have looked at the throat we have seen there a white superficial exudation on the tonsil that we recognised at once as syphilitic, and we knew that we had to deal with an exceptional case of acute febrile outburst in connection with secondary syphilis. On listening to the chest we may hear bronchial râles, showing that there is syphilitic inflammation of the bronchial tubes, which may even have passed into broncho-pneumonia. We may at the same time detect albumen in the urine, and we have then to ask ourselves whether this albuminuria is merely due to venous congestion, such as we may meet with in various febrile conditions, or in weak states of the body, such as this of syphilitic cachexia, or if the albuminuria indicates nephritis. Should we send the patient to bed for days or even weeks, or should we let him go on with his work? In this connection I always think of three cases I have seen in medical men, all of them cases of infection from midwifery practice, in which this question came before me, whether I should send the patient to bed, or let him go on with his practice. One of them went on with his work as usual, but of the other two, one had albumen in his urine, and the other had albuminuria and œdema, and I had to keep them in bed for some time. That nephritis may occur during the secondary stage of syphilis is beyond question, and the cases vary very much in severity. More commonly only a small area of the kidney is affected, and we need not take the patient away from his work; but in other cases the disease may spread through both kidneys, and the patient may die from uræmia. In considering the question of keeping such cases in bed, we have to remember that when a patient is going through this syphilitic trouble he is usually much depressed; that I noticed in all those medical men. One of them had prolonged depression, and eventually he had cancer and it killed him. So that in view of that depression we must not send such patients to bed unless it is indispensable, and the same may be said of the lung condition. However, the changes of secondary syphilis very rarely kill; if the patient dies it is usually due to some collateral disease. What we think of when we speak of internal syphilis, and what we see on the post-mortem table, are chiefly congenital and tertiary syphilis.

To give you a summary of the changes produced by tertiary syphilis, I think that I may say that it may attack any part of the body, but it most commonly affects the liver; nearly as commonly as the liver it affects the testicle; viscera often affected are, the brain, the lung, and the kidney. Exceedingly common is syphilitic disease of the periosteum and bone. Tertiary syphilis may affect the spinal cord, and the spinal as well as the cranial nerves. Syphilitic disease of the arteries is very common. The most severe and most extensive gangrene I have ever seen was in tertiary syphilis, gangrene of the extremities in connection with tertiary syphilitic disease of the liver and other parts. The vessels of the lung may be affected, and gangrene may ensue. I have seen a peculiar sloughy condition of the tonsils from a similar cause. Syphilitic disease of the spleen is rare; it is associated with liver disease. I have no case in my mind of syphilitic disease of the stomach, but syphilitic disease of the rectum is not uncommon, leading to stricture. I have also seen one or two cases in which tertiary syphilitic ulceration of the intestine led to death by setting up protracted diarrhœa. In passing, I may allude to the fact that changes in the eye are not uncommon. The tongue also very commonly suffers.

I must refer rather more fully to the changes in the brain. Syphilitic subacute inflammation may lead to increasing thickening of the membranes of the brain, extending into the vessels of the convolutions, leading to increasing dementia, a mindless condition, which continues until the patient dies. With the mind failure there are commonly recurring fits, and much pain in the head, which is worse at night. Syphilitic gummous changes may occur in the corpora striata and optic thalami, and produce hemiplegia with comparatively little disturbance in the mind; but such cases are rare. If we are asked about syphilitic disease of the brain we must immediately think of surface changes, because these are by far the most common. The same applies to the spinal cord. Syphilitic paraplegia comes on with aching pain in the limbs, which is commonly thought to be rheumatism. After a time weakness is noticed in walking. There may be weakness in the arms also, but more commonly it is confined to the lower extremities. The difficulty in walking increases until there is decided paraplegia. If there is myelitis in addition to the syphilitic thickening of the meninges, there may be more or less loss of power over the bladder. These symptoms commonly disappear with rest, or with the ordinary mercury or iodine treatment, and the patient walks about again, but there is liability to recurrence. What we have to recognise with regard to this recurrence is this, that there is a small gummous change in the pia mater of the cord, and there is also fibroid change, which has to some extent spread into the connective tissue of the cord. In that connective tissue lie the blood-vessels, arterioles, and capillaries, and these vessels are weakened by the fibroid thickening; they are liable to get over-distended, and inflammatory exudations pour out; myelitis comes on. Further, the fibroid tissue contracts if it is in a more advanced stage, and constricts the nerve-tubes. Thus we may account for the characteristically recurring myelitis in these syphilitic conditions, and can understand that if the tissue changes are very extreme there may be permanent paraplegia.

What are the appearances of syphilitic disease in infancy? We notice a little tubercular-looking swelling about the anus, coppery in colour, and there is also some fissuring of the skin. We notice a similar superficial ulceration or cracking about the angles of the mouth, and the skin about there is also of a coppery red. On inquiry we learn that the child snuffles a good deal, and further we are told that it was a fine child when born, but that latterly it has been wasting. Putting all these things together, we have no doubt that it is infantile syphilis. I do not, however, purpose to dwell on these infantile conditions.

Before closing this lecture I wish more particularly to call your attention to syphilitic disease of the rectum. On looking at the anus, we notice that the skin in its neighbourhood has no longer the healthy pink look, but has a peculiar dull white fibroid appearance. This fibroid thickening, radiating from the anus over the buttock, should at once lead us to think of syphilis. On passing the finger into the bowel, we find that the fibroid thickening extends through the sphincter, and that the wall of the lower part of the rectum is much thickened and indurated. We may next notice that the mucous membrane is destroyed in one part—in fact, there is an ulcer with well-defined edges—so that we say at once that it has the characters of a tertiary syphilitic ulcer. Around the bowel in the neighbourhood of the ulcer is a dense mass of fibroid thickening which has contracted, and constricted the lower part of the bowel. The question commonly arises in these cases whether the disease is scirrhus or syphilis. Scirrhus also forms a dense fibroid mass in this region, and causes a similar constriction of the bowel, but the constriction is more extreme than that due to syphilis. In making post-mortem examinations I have been impressed with the fact that in scirrhus there is a more circumscribed annular constriction, not such a large dense mass as we find in syphilis. The fibroid thickening of the skin around the anus is a further distinction of the syphilitic cases. The presence of the tertiary ulcer in the rectum is a further guide, and we may find signs of tertiary syphilis in other parts of the body. In these cases the symptoms recur on and off for years, whereas in cancer the case progresses steadily until death.

On cutting into such a mass in cases in which syphilitic stricture had caused death by obstruction of the bowel, I have seen a tertiary ulcer surrounded by much dense fibroid substance, and large quantities of fat were intermixed, so that the whole formed a large hard tumour in which the rectum was imbedded; and, as I told you in speaking of the liver, so here, in some cases there is very little sign of gummous material, whilst in others we find a large quantity. Syphilitic disease in the upper part of the intestine is so rare that you need only remember that there may be one or more tertiary ulcers similar to those that occur in the skin.

I must draw your attention to one more organ that is subject to syphilitic changes, and that is the heart. As in the liver, the connective tissue of the left ventricle may be thickened by fibroid growth. A dense tough fibroid growth radiates through the muscle in the form of a network, or we may find one dense fibroid mass about half an inch in diameter, or there may be dark congested gummous masses scattered through the wall of the left ventricle. All of these changes alike lead to dilatation of the ventricle. I remember also seeing a case in which the dark gummous masses took on acute changes and sudden death occurred.

LECTURE XXI.

LARDACEOUS DISEASE.

The three common antecedents of lardaceous disease are tertiary syphilis, tubercular and strumous changes, and protracted suppuration. Lardaceous disease may follow prolonged suppuration whatever its cause, but as the commonest cause of protracted suppuration is bone-disease, we more often see lardaceous disease in surgical than in medical wards. If we find evidence of lardaceous disease in the liver, spleen, and kidneys—these are its most common seats—we should always look for signs of syphilis, phthisis, or bone-disease. I know nothing of lardaceous disease occurring as an idiopathic affection; when I have met with it there has been always one of those recognised antecedents.

What is the kind of suffering that occurs with lardaceous disease? First, there are symptoms of one or other of those conditions I have mentioned to you, phthisis or other signs of struma, syphilis, or bone-disease. The patient has been ill for months, or it may be for years, and we then notice that the liver or spleen, or both, are becoming enlarged. There is a regular smooth enlargement, the consistence of the organs is very firm, and the edge is very sharply defined, and these characters lead us to feel sure that lardaceous disease has attacked them. As the enlargement of the liver and spleen progresses there is increasing wasting of muscle, and commonly diarrhœa comes on, and we are led to think that lardaceous material is being deposited in the mucous membrane of the intestine, and that this is leading to the recurring diarrhœa, and with that to increasing wasting and increasing pallor. About the same time we may find albumen in the urine. There is usually much albumen, and the quantity of urine is in excess of the normal daily average; but the urine is not much altered in colour, nor is there any constant change in the specific gravity, but with the microscope we find tube casts. As the disease of the kidney advances there may be loss of colour in the urine. The kidney changes may lead to the production of some œdema, but not extreme œdema such as we see in cases of large white or mottled kidney. The enlargement of the liver and spleen is sometimes very great. If you are asked what are the largest livers we find on post-mortem examination, you should answer, lardaceous livers, and livers with scattered medullary cancer. With the great enlargement of the liver, ascites comes on, and then we see a very impressive sight, typical of lardaceous disease: the belly is big, the veins over the abdomen are greatly distended, the liver and spleen bulging at the top, and, adding to the general enlargement, fluctuation very distinct, the skin extremely pale, the legs and arms extremely emaciated, the cheeks and eyeballs sunken; the whole appearance is so characteristic that when we see it we at once suspect lardaceous disease, the result of bone-disease, for phthisis usually kills before it gets to this extreme. The emaciation is brought about by the recurring diarrhœa, and by the catarrhal condition of the mucous membranes; the tongue is extremely red and parched, beefy-looking, and shines in a way that leads us to suspect that there is deposit of lardaceous material in it.

It is very remarkable how many years some of these lardaceous cases may continue. A man may have had tertiary syphilis; enlargement of the liver and spleen, and albuminuria, may come on; there is undoubted lardaceous disease, and these symptoms may be more or less severe for twenty years. I had a patient who was said by Dr. Budd, twenty years ago, to have lardaceous disease. Many times I have examined that man since; he is living still, he is an engineer in the city. In some cases the symptoms may entirely disappear, so that we are led to think that lardaceous disease is a curable affection. But if the antecedent disease is increasing in severity, if, for example, there is advancing bone-disease or progressive phthisis, then the lardaceous disease increases and kills the patient.

What are the morbid appearances of lardaceous disease of the liver, spleen, and kidneys? The liver is extremely large, thick, and heavy. It does not feel tough like a fibroid liver, nor nodular like cancer, but as if it were uniformly infiltrated by a dense, waxy material. The surface is smooth and shiny. On section we notice that it cuts like suet, and we observe that the lobular structure is for the most part lost. In place of it is a fine granular, almost homogeneous, infiltration through the substance, of a dull greyish-red colour, which we immediately recognise as due to lardaceous disease. On pouring iodine over the cut surface it stains a deep mahogany brown, whereas the healthy liver similarly treated remains the pale brown colour of iodine itself. There are two varieties of lardaceous affection of the spleen—a uniform infiltration, and what is known as "sago-spleen"; but as I shall describe these when I come to lecture on diseases of the spleen I need not dwell upon the matter here. The kidneys are usually much enlarged; some of the largest kidneys are those which are the seat of the lardaceous change; the capsule is consequently much stretched, and the kidney has a grey waxy-looking, shiny appearance, with congestion here and there, leading to a somewhat mottled appearance, but the shining look is very different from that of the large white kidney of Bright's disease. On cutting into the kidney we find the cortex much swelled by the waxy substance, and the outline of the cones is hidden by the deposit. The iodine test applied to the kidney is not so successful as in the case of the liver and spleen, but with the microscope we get a better result. We see the Malpighian bodies of a dark brown colour, and the tubules are also more or less affected.

The microscopic appearances of the kidneys in this disease are very striking. If we do not know them we are puzzled by seeing the structures so sharply defined; the convoluted tubules, the Malpighian bodies, and the vessels stand out sharply; but with this increase of definition we notice an extremely granular, cloudy condition of the epithelium and tubules that tells us at once there is waxy degeneration. In the liver we notice that the liver ducts and the liver cells look unusually definite, but there is a hazy substance, extremely finely granular, in fact almost homogeneous, diffused through all the structures more or less, especially the epithelial. In the spleen we see the same finely granular, not quite homogeneous, cloudy infiltration, and we find the same in the mucous membrane of the ileum. Muscle also is sometimes affected by the lardaceous change.

At one time it was thought that the infiltrating material belonged to the class of amyloid bodies, but that view has been abandoned, and chemists regard it as a nitrogenous compound, a modified albumen, that has originated from a weaker and lower form of nutrition.

LECTURE XXII.

DIABETES.

I WILL introduce this subject by saying to you that in the most virulent diseases, in the most malignant forms of disease, we find the least morbid appearances after death. I have called your attention to this fact in speaking of rheumatic fever, of malaria, of cholera, of small-pox; it applies equally to diabetes.

If you are asked to describe the morbid appearances in death by diabetes, always say, "Do you mean acute diabetes or chronic diabetes"? Ask that question pointedly. In death from acute diabetes we may find no morbid appearances which will warrant us in coming to a conclusion as to the cause of death. There may be a sweet smell, like that of new hay, or some other sweet smell, which may make us suspect diabetes; but from the morbid appearances alone we cannot positively say it has been a case of diabetes, for similar morbid appearances are found after death by epilepsy, apoplexy, or typhus fever. We could not go into the witness-box and swear the death was due to diabetes. You may be asked why you call diabetes a definite disease, since there are no morbid appearances characteristic of it. Answer, that the clinical course of acute diabetes is absolutely characteristic.

A patient has the early symptoms of diabetes. He passes an excessive quantity of urine with a high specific gravity, containing much sugar; there is great thirst, loss of strength, and some wasting; we examine the lungs carefully and find no objective signs of disease, and we say it is diabetes. We can obtain no evidence of any organic nervous lesion, there are no signs of liver cirrhosis, of syphilis, or of phthisis, but the patient becomes rapidly weaker. He goes about much as usual, whilst not thought to be extremely ill, certainly not thought to be dangerously ill, but he becomes rapidly weaker; perhaps a great deal of emotional disturbance comes on. There is increasing tendency to stupor, the circulation rapidly fails, the pulse becomes smaller and smaller; the stupor increases, the face becomes congested, and is red rather than blue; the hands feel cold; the skin, which hitherto has been peculiarly dry, becomes covered with a cold sweat; the breathing becomes labouring, and it is evident that the circulation and the breathing must shortly cease. Patients may lie for many hours in that condition, with no pulse at the wrist, with labouring inspiratory movements, excessive action of the diaphragm, completely comatose, and with a peculiar red appearance of the face.

Now, when we make the post-mortem examination, what do we find? Venous congestion—nothing more. Venous congestion of brain, liver, kidneys, stomach. When we examine the lungs, all we can say about them is that they are congested; the congestion may be extreme, so that they weigh very heavy, and when we cut into them we find that they are black with accumulated blood; in other cases they weigh much less than normal, but have a dark red venous appearance. There is nothing characteristic about these morbid appearances, which are found in death from various causes.

It is supposed that in these cases the sugar which has accumulated in the blood and the tissues undergoes decomposition and poisons the patient—acetonæmia it has been called—but this is pure theory. What we have been brought to think about it is that there is some great failure of the cerebro-spinal system antecedent to the diabetes. We think that it is the increasing failure of the nervous system which leads to rapid death from coma, and not the so-called diabetic condition. In treating diabetes I have come to consider especially the nervous condition and to look out for danger in the nervous system.

In other cases of diabetes the diabetic symptoms may last for months, and then symptoms of pulmonary disturbance supervene. In the more acute forms we often meet with gangrene of the lung. There is cough, shortness of breath, and expectoration. We notice that the latter becomes very offensive, and day after day the patient goes on expectorating a dirty brown material, or it may be greyish like mortar, and it has a very offensive smell. The patch of gangrene may be so small that we fail to detect it by physical signs ; and even though large, if it is deep in the lung, the only evidence of gangrene will be derived from the general symptoms. This condition may end fatally, or in recovery.

Instead of pulmonary disturbance there may be inflammation of the skin. Where we find much exudation into the skin, not affecting merely the surface, but extending deeply, and the inflammation goes on increasing in spite of treatment, we should always examine the urine for sugar, for such inflammation of the skin is very common in diabetes. The inflammation may go on extending for several days, and then we may find several foci of suppuration, and we know that we have to deal with a carbuncle. Now these large carbuncles in diabetes sometimes kill a patient very rapidly. I want to impress this point on you, that any inflammation in a diabetic patient is always attended with very great risk to life ; but in some cases they recover from such troubles, and the symptoms go on much as before.

The patient may be attacked rather suddenly with shortness of breath, fever, rapid pulse, exceedingly viscid expectoration, and all the other symptoms of acute pneumonia ; at the post-mortem examination we find the ordinary appearances of red hepatisation, or the lung may be passing into grey hepatisation, and there may even be purulent infiltration ; most of the cases I have seen have shown red hepatisation, with a little grey hepatisation. You will bear in mind that hitherto I have been speaking of acute diabetes, that all the incidents I have described are liable to occur in the early stages of the disease ; in chronic diabetes we find very different conditions of the lungs.

In considering the pathology of diabetes it has been usual to look upon the disease as consisting mainly in the passing of sugar in the urine, to take a symptomatic view, and to follow a symptomatic plan of treatment. Physicians have looked only at the outward sign, that day after day they could find sugar in the urine. As the patient became weaker and weaker more attention was paid to the sugar, for it was considered that the passing of such quantities of sugar was causing the weakness of the patient, and the treatment was more vigorously pushed.

Now, in studying pathology we have always to ask ourselves this question, How can we look at the disease in a way to get more success in treatment? As soon as I find a view does not help me to treatment I am quite willing to give it away to some one else, but I have no wish to keep it myself.

Let us ask what has been the success of this symptomatic pathology and treatment of diabetes. The patient certainly came very often to see the doctor, and there was a success in that; but sooner or later the patient died of the disease, and many people were satisfied, and there was success in that. Now I do not want you to think that such pathology and such a method of treatment were of no use at all, but I want you to look facts in the face, and see where their use lay. Year after year I have made post-mortems in this hospital, and heard the same story: "This is a case of diabetes. The patient was passing much water of high specific gravity, containing a large quantity of sugar, and we put him on a strict diet; we gave him gluten bread, we cut off potatoes, and the quantity of sugar passed was less, and the specific gravity of the urine diminished, and the patient seemed to be improving." "Yes; and there lies his body." I have heard that story from one house-physician after another. They said that they had dieted them very strictly indeed, and the sugar had entirely disappeared—but there was the body.

Then it came home to me very clearly that treating the patient so that he ceases to pass sugar does not necessarily cure him; he may not pass sugar for six months before he dies, and yet all that time be getting nearer and nearer death.

I told you that the patient might die rapidly, and after death no morbid appearances be found that we could definitely attribute to diabetes. The sugar may, after we have detected it, soon be passed in much smaller quantities, or the patient may go on passing a considerable amount for years; it may then disappear from the urine altogether, leaving the patient much stronger, but not entirely well.

You see that we have to look beyond the mere presence of sugar in the urine; let us follow the suffering. When the severity of diabetes is increasing there is increasing weakness, increasing thirst, more urine passed (it may be ten or more pints daily), the specific gravity is high, commonly between 1030 and 1040; the skin becomes drier and drier, the patient becomes mentally more and more depressed, his appetite becomes ravenous, and yet he derives less and less comfort from food; he is ever hungrier, and ever enjoys his meals less; his nights are much disturbed. The patient will often say, "I suffer so terribly from thirst, but I do not find that any quantity of water will slake my thirst—in fact, I find that the more I drink the thirstier I get; I would rather not drink so much." This makes us think that the thirst is due to some disturbance in the nervous system, and we find that a little opium gives much relief. But sometimes the thirst will become less without treatment. Only this morning I saw a young woman who had been suffering terribly from thirst. I had ordered her some Dover's powder, but she had not taken any of it. She said to me, "My thirst is much less, but I have such a burning sensation in the mouth." The patients themselves have taught me that there is some paræsthesia in the mouth. No doubt the system demands more water, but there is a nervous disorder which prevents satisfaction of the thirst in the ordinary way.

In diabetes there is much suffering from constipation, for the fæces have not enough water to keep them soft, the mucous membranes, like the skin, being dry and hard; the parched skin is often reddened, and is liable to blotchy eruptions on it. As I told you above, there may be more serious failure of function, leading to severe inflammation. In the dry mouth catarrhal inflammation is apt to arise, the surface is covered with a sticky mucus, and in this spores are apt to settle, and there is a rapid formation of the thrush fungus in the decaying mucous membrane. There may be an enormous growth of mycelium in the pharynx and œsophagus, so that at the post-mortem examination we can scrape away spoonfuls of it. In these cases there is great danger of the catarrhal inflammation spreading down the bronchial tubes, and this is enough to kill the patient; but it is only the inflammatory process which extends into the air-passages; the growth of fungus is limited to the mouth, pharynx, and œsophagus.

There is a similar catarrhal inflammation of the urinary passages, and in women this may involve the whole vulva, which causes a good deal of distress, burning and itching, with a purulent discharge both from the vulva and vagina; in the male the urethra is affected, with smarting and itching at the end of the penis. If a patient comes to us complaining of excessive irritation of the genitals, we should always examine the urine for sugar.

After these symptoms have continued for months with varying intensity, we may notice another sign. We find that the breath-sounds at the apex of the lung are no longer vesicular in character; they get harsher and harsher, more and more tubular; we notice that there is sinking in of the supra-clavicular and infra-clavicular spaces, and that the percussion note is getting flat. Soon we notice a duller note at one apex than at the other, and now and then we can detect a little moist râle; but there is very little cough and very little expectoration; the progress is exceedingly insidious. Next we have similar signs at the other apex, and the râles become louder and more distinct, so that we have no hesitation in saying that there are phthisical-like changes in the lungs. I will tell you why I use that indefinite expression. It has been a question on which distinguished pathologists have held different views, as to whether these changes in the lungs of diabetic patients are precisely similar to those found in cases of phthisis. My own experience is that the changes are not usually tubercular, but that they are due to catarrhal pneumonia; there are small patches of inflammation here and there. As the lung function fails, as the lung tissue wastes more and more, catarrhal pneumonia comes on, and gradually creeps through the lung; in exceptional cases I have been unable to distinguish the morbid changes from those of phthisis, and therefore I think there is right on both sides of the dispute. Cavities form, and they may be so large that when the lungs are put before us we say at once that there has been phthisis, but we add that we should like to know what the antecedents of the case have been. I will explain why I put it like that to you. I know no morbid condition, no chronic morbid condition, which may not in its extreme stage present phthisical-like changes in the lungs. I have seen such changes in cancer, in valvular disease of the heart, and in every kind of chronic disease; hence, when we find phthisical changes in the lungs, we must always, before dismissing the case as one of pure phthisis, ask what the antecedents have been.

Let me give you now a summary answer to the question, What are the morbid appearances in the lungs usually met with in cases of diabetes? In acute cases there may be simple congestion; the congestion may be attended with more or less red hepatisation; there may be also scattered patches of grey hepatisation; we may find gangrene of the lung. Again in other cases some of the consolidation has broken down and formed cavities like those of acute pneumonic phthisis—galloping consumption. In other cases the masses of pneumonic consolidation have been small, from the size of a pea up to that of a large walnut, and many of them have undergone caseous degeneration and softened down into cavities; in other cases, in addition to the cavities, we find towards the bases of the lungs tubercular changes like those met with in ordinary tubercular phthisis. If asked whether you mean miliary tubercle, say, No: that you mean the tubercular changes which are met with in connection with pneumonic phthisis, boiled-rice tubercle, not the grey granulations seen in acute tuberculosis.

In an ordinary case of chronic diabetes this disorganisation of the lung extends and the heart's action becomes exceedingly feeble, until finally the patient succumbs to some trifling acute inflammation, as a little pleurisy, a little pneumonia, or, as I told you, thrush. If there is much wasting of the body, and in the end it usually becomes much emaciated, we notice also signs of atrophy of the brain. I have been brought to think that the disorganisation of the lungs is merely the ultimate effect of the failure of nutrition in which the disease we call diabetes consists. The sugar in the urine, the wasting of muscle, the disorganisation of the lungs, are all outward manifestations, dead effects, but not the disease itself. If we want a view which will help us to treatment we must look further afield. When the ship is on the rocks and is going to pieces, lowering the boats does not help us to get her off and into sailing order again ; but we must try to prevent the next ship from going on the rocks.

I saw a young man with diabetes a little while ago, and when he was stripped I could see that he had been an active, vigorous man. I asked him what he thought had been the cause of his illness, whether it could be the close air of the Clearing House in which he worked. He said he did not think it could be that, as he had been working there for many years, and had never felt it affect him, but he thought that it was due to excessive excitement and exertion in swimming competitions. He did not look like a man who had given way to excesses of any kind, except in the way of activity. I remember another case of a young woman, whose father had died of diabetes, and who was herself the subject of it ; she said she had suffered extremely from nervousness so that she was unable to speak at times. I remember a young man who, whilst very actively employed, had a nervous disorder for which he used to come and see me—I could not give it any definite name ; but he ultimately got brain failure and hemiplegia, and died of diabetes. The more I study the question the more I am convinced that there is a nervous disturbance antecedent to diabetes.

Let us examine the question from the other side. It is beyond question that in various organic diseases of the brain we find sugar in the urine. Syphilitic disease of the brain, for instance, may be attended by sugar in the urine, and symptoms leading us to think of diabetes. Further, where there has been failure of general nutrition, as in cirrhosis of the liver, we may meet with sugar in the urine, and we find the same thing after great anxiety. In some cases of emphysema with arterial degeneration and dilatation of the left ventricle, progressing from year to year, we find sugar in the urine. When sugar appears in the urine the body wastes, the skin becomes dry, and there is thirst. Just when we have agreed to call it diabetes, and the sugar disappears from the urine, there is an attack of cerebral hæmorrhage. Dr. Savage has asked me, "How should you account for this, that a patient may have diabetes, and when there is a maniacal outburst, the sugar often disappears from the urine; and further, that as the mind recovers, the sugar is again found?"

I cannot define the nervous disturbance—it is something peculiar, and at present beyond our grasp—but I am ever more impressed with this, that diabetes is not a mere disturbance of glycogenesis, but is preceded by some disturbance in nervous function.

LECTURE XXIII.

DISEASES OF THE LARYNX.

You will remember that I have taken up the old distinction that disease means that the easy working of the part is interfered with. We see this very clearly when we have to deal with diseases of the larynx. You will find many illustrations to show that the larynx is much affected by mental diseases. If you talk to a very nervous person in a strained harassing way, often a nervous spasmodic condition is produced in his larynx which reveals itself by a tickling harassing cough. What we mean by worry is hindrance. If a person comes and worries us we cannot get on, for there is an untimely interference with natural functions. If such worry and interference become still greater, instead of spasm of the larynx there may be a paretic condition, and the voice is reduced to such an extent that we can scarcely hear it; this is the so-called hysterical voice, the aphonia which is common in conditions of nervous exhaustion. But before discussing this I will speak of other conditions which lead to aphonia.

What is aphonia? It is a soundless condition of the larynx. It comes on very commonly when the mucous membrane of the larynx is swelled, and therefore a common cold in the throat is attended by hoarseness. It may be caused by growths on the vocal cords. The vocal cords, like the skin, are subject to warty growths or papillomata; these growths consist of fibrous tissue thickly covered with epithelium. Aphonia may also be produced by inflammatory conditions which have extended to the larynx from below, and we must not overlook that huskiness may be due to some severe disease of the chest, such as an aneurysm pressing on the trachea; or there may be emphysema with bronchitis, and extension of the inflammation upwards. There is a typical aphonia due to aneurysm pressing on the recurrent laryngeal nerve. The tension of the cords is no longer normal, and there is a rough, low-pitched, broken voice, and the cough is gruff and clanging. With the laryngoscope we can see that the abductor action of one of the cords is paralysed. If it is the left, the aneurysm must spring from the arch of the aorta; if it is the right, the aneurysm probably arises from the first portion of the right subclavian. Paralysis of the abductor movements of both vocal cords is almost always due to disease of the central nervous system. We get aphonia in nervous diseases. It may be almost the first sign of degeneration of the spinal cord. It may be the first symptom, or rather one of the first symptoms, of general paralysis of the insane. It is part of the complex of symptoms known as labio-glosso-laryngeal paralysis. We see it in lateral sclerosis, in locomotor ataxy, and in sclerosis of the brain coming on with increasing dementia. I have often told you that spasm and paralysis are akin, and as an instance of this we see sometimes a spasmodic affection of the larynx as an early symptom of general paralysis of the insane. A young man used to come to me at the out-patient department who said he had whooping-cough. He suffered from spasmodic attacks of laryngeal cough. I did not then consider the case to be serious, but he died in hospital about twelve months later of general paralysis of the insane.

Aphonia occurs in so-called hysteria. What is the condition of the larynx in hysterical aphonia? With the laryngoscope you can see that the vocal cords when they should come together for the act of phonation act lazily. When you see this lazy movement you say, "I'll whip them up," and you go and get a battery and do it, and the patient speaks. Then you are very pleased with yourself, and say, "I did it!" But in a few days the patient comes back again as bad as ever. You "whip them up" again. Three months pass. You were quite sure you were going to cure the patient, but when you find at the end of three months that she is no better than she was at the beginning you say there is a partial paralysis of the vocal cords. That sounds well. I have often seen this go on for months and months, and at last the doctor gave it up; the patient beat him and he got tired of it. A young girl had hysterical aphonia, and I had not time to use the battery. I took up a volume of Shakespeare and read a little to her, and then I handed her the book saying, "Now you read to me," and she did it. I did not give her time to think. This condition arises from a tired mind, and you cannot cure a tired mind with a battery.

It was the contention of a very noted German writer that thinking is silent speech, and that when we are thinking a great deal more is going on in the throat than we are aware of. If we try to think earnestly while in the act of swallowing, very likely some of the food will pass into the larynx. You will often read in the papers of cases in which people have died of choking while eating. These are generally cases in which the speech organs have to some extent failed, and there is some anæsthesia of the upper part of the larynx, often in old people who are somewhat demented—thinking while swallowing is the cause of the passage of food into the larynx.

Aphonia in hysterical patients is associated with more or less anæsthesia. In various melancholic conditions loss of voice is common. Aphonia comes on in collapse—in severe cases the voice is reduced to a mere whisper, as in cholera, in perforation of the intestine, and in bad accidents.

If the larynx is much overworked the automatic function begins to be interfered with, the vessels get weak, the mucous membrane is relaxed, and we have to deal with what is known as "clergyman's sore throat" common in clergymen, public speakers, and singers. I once had to treat a case of this kind in a bookmaker who had to shout on a racecourse. Owing to the failure of the nervous regulation of the larynx and the weakened condition of the vessels, catarrhal inflammation is apt to come on, and to recur. In the worst cases the epithelium is shed and the mucous membrane becomes granular, and the disease may last for months and even years.

Let us go on to consider catarrhal inflammation of the larynx from other causes. Most commonly it is due to exposure to wet and cold in the winter months; but it occurs also severely in persons who are not so exposed but whose nutrition is weak, very sensitive delicate people, and they may contract these catarrhal conditions without leaving the house, even without leaving bed. You have to remember how different are the susceptibilities of different people, and that the effect of the east wind on different individuals varies much.

Catarrhal inflammation of the larynx is very common in anæmic conditions, and anæmia is very common in strumous conditions. We can perhaps best put it in this way: Anæmic people, with a tendency to phthisis, are very liable to catarrhal laryngitis. Catarrh of the larynx tends to come on when breathing is weak, and therefore it is very common with vesicular emphysema of the lungs. In emphysematous persons an inflammation may begin in the bronchi and extend up to the larnyx; or, conversely, may begin in the larynx and, by extension, set up bronchitis. Catarrh of the pharynx also may extend to the larynx. It is important to bear these facts in mind, because in some cases of emphysema there is much coughing and wheezing, and the sounds are transmitted to the chest-wall, and it is difficult, as I have myself experienced, to say whether the bronchial tubes are or are not affected. We have to recognise that the catarrhal inflammation in such cases may be much marked in the larynx, leading to difficulty of breathing, and in some cases a good deal of laryngeal spasm may come on, causing asthmatic attacks. We have to remember, then, that in emphysema, dyspnœa and asthmatic attacks may sometimes be due to laryngeal conditions, not to bronchial. In some of these cases the patients completely lose their voices, and this is a great trouble to them. The remedy for these laryngeal asthmatic seizures is the application of a very hot wet sponge over the larynx, so as to soothe the circulatory and nervous working of the larynx. You would not hesitate to give a little opium.

A similar form of catarrhal laryngitis is apt to come on when there are phthisical changes in the lungs. We have to recognise that there are different forms of phthisical change in the larynx. In the simplest form there is catarrhal inflammation, and this leads to much coughing, and, it may be, to almost complete aphonia. Such catarrhal inflammation of the windpipe may occur winter after winter, until at last there is phthisis; and this is the reason why people who are going into phthisis say, "I am always catching cold." So the first thing we may notice in strumous disease of the larynx is this—the mucous membrane is congested. The congestion spreads over the arytenoid fold, the epiglottis, and the vocal cords, and it tends to soften the parts, so that function is rendered exceedingly difficult. If it goes further, the inflammation leads to destruction of the epithelium, which then becomes very granular, and the mucous membrane is swelled by an albuminous exudation. This condition passes into superficial ulceration, which is always most marked over the posterior parts of the vocal cords. It is here that we look for the early ulceration of larnygeal phthisis. As the condition advances, the mucous membrane becomes more swelled, granular, and gelatinous-looking; this thickening and gelatinous appearance is most marked over the summit of the arytenoid cartilages and in the aryteno-epiglottidean folds, so that it forms a swelled band around the entrance to the larynx, which makes it difficult to see the vocal cords with the laryngoscope. Ulceration takes place in that gelatinous tissue, and, in the most destructive forms, the arytenoid cartilages, the vocal cords, and much of the epiglottis are eaten away. We are liable to mistake this condition of extreme ulceration for a result of syphilis. Where I have found such changes in the larynx I have found evidence of phthisis in the lungs; but at the outset the morbid condition may be so much more marked in the larynx, and the symptoms may be so predominantly laryngeal, that we are warranted in speaking of it as laryngeal phthisis. But I have never yet seen a case in which I could say that the phthisical change was limited entirely to the larynx.

Another condition causing general venous congestion, and therefore not uncommonly associated with catarrhal laryngitis, is heart disease. A patient comes to the doctor and says, "I am troubled with such a harassing cough; it comes on with such fits, and it nearly suffocates me." When you hear this, never fail to examine the chest, for it may be that an aneurysm is beginning to press on the trachea. And another thing which may lead to such a cough as that is cancer at the root of the lung. I saw a case in a medical man, in which the patient himself at first thought that he was suffering from whooping-cough, but he died more than a year later of intra-thoracic tumour, which had grown upwards along the trachea.

Here is another case. A man had catarrhal laryngitis, and was treated by Sir George Burrows and myself. He got no better, and I suggested that he should see Dr. Walsh. The latter said to me, "As soon as I heard him cough I said to myself that he had an aneurysm pressing on his trachea." Then I took the patient to see Sir William Gull. He said, "It is not aneurysm; the laryngitis is due to disease of the spinal cord." Now here were these two men, who had had such immense experience, and they differed as absolutely as this. I must confess that when I heard Sir William Gull say it was disease of the spinal cord I was much surprised. The man lost his reason, and he died.

You see that I have good reason for putting before you that catarrhal inflammation of the larynx and trachea may arise in several ways, and that in some cases its origin is very obscure; but when it is severe and repeated, and resists all remedies, we may feel confident that there is some deep-seated cause for it.

Another morbid condition which may give rise to pressure on the trachea is enlargement of the thyroid gland. This gland undergoes simple hypertrophy, and it grows under the sternum, and, enlarging behind the sternum, it compresses the trachea, and produces spasmodic attacks of dyspnœa, very similar to those produced by aneurysm.

The trachea is very little subject to primary disease; in fact, such disease, excluding syphilis, is almost unknown, and disease of the trachea is commonly due to pressure from without, or to extension of inflammation from above or below. In very rare cases we meet with syphilitic stricture of the trachea, causing symptoms almost indistinguishable from those which arise from an aneurysm pressing on the bifurcation of the trachea. In these cases we have the usual signs of tracheal narrowing, but we cannot feel an enlarged thyroid, nor can we detect aneurysm nor signs of intra-thoracic new growth. Post-mortem, we find an annular fibroid mass in the wall of the trachea, and in the mucous membrane a gummous formation, of the colour of wash-leather. The contraction of the fibroid tissue has narrowed the trachea, and the patient may have died in an attack of dyspnœa, or the persistent irritation may have set up a suppurative inflammation, which has travelled to the lungs.

I will sum up the diagnosis of these conditions as follows: Whenever there are severe and repeated attacks of dyspnœa, and there are no obvious signs of organic disease to account for it, we should think of pressure upon the trachea or one of the larger bronchi. This view is supported if there are signs of catarrh in those parts, wheezing, a sensation of clogging phlegm, and the coughing up of ropy mucus. Sooner or later we may get distinctive signs that the passage is narrowed, in the development of the harsh tubular sound as the patient breathes, that is so characteristic of constriction of this part. Then comes the question, What is the cause of the narrowing? We have to consider the possibility of cancer, of aneurysm, of enlarged thyroid, and of syphilitic stricture. Any gross enlargement of the thyroid gland is easily detected. Failing that, and if we cannot find any other signs of intra-thoracic aneurysm or of cancer, the case is probably one of syphilitic stricture. I must repeat, however, that this condition is exceedingly rare.

What are the more common forms of syphilitic disease of the larynx? In its early stage we cannot distinguish it from catarrh of the larynx. The voice is husky, there is troublesome cough, and expectoration of mucus. But what rivets our attention is the persistency of the inflammation. What, we wonder, can be the cause of this persistent catarrh of the larynx? A history of syphilis, or the co-existence of other tertiary manifestations, lead us to suspect that the laryngeal affection is syphilitic. As the inflammation continues there is an accumulation of inflammatory material in the aryteno-epiglottidean folds, on the surface of the epiglottis, and the upper part of the larynx, the accumulation being always most marked between the epiglottis and the true cords. As is common in tertiary syphilis, the inflammatory material undergoes fibroid thickening; tough, thick, whitish fibroid tissue is formed; but, as a rule, in the larynx we find little or no evidence of gummous growth. In the thickened tissues ulceration is prone to occur. I have seen a single tertiary ulcer, similar to those we find in the skin; but more commonly there is a scattered ulceration, by which much of the vocal cords is eaten away, and by which the epiglottis is especially affected. It is characteristic of syphilitic disease of the larynx, as of the brain and other parts, that the ulceration heals, and then recurs, but after some months again heals, to break out anew, and in this way the disease may continue for years. The fibroid material in this part, as in others, tends to contract and to constrict the upper part of the larynx. Whilst these processes are going on, the danger lies in the inflammation travelling down the trachea and bronchi to the lung substance, and setting up fatal broncho-pneumonia.

Recognising that risk, and seeing that the dyspnœa is becoming more severe, the question comes, shall tracheotomy be performed? What do we expect to gain from the operation? The operation may be done for one of two reasons—firstly, it may be done to ward off immediate death, because the attacks of dyspnœa have become exceedingly violent, and the bronchial tubes are getting choked by muco-purulent matter; the other reason for which tracheotomy has often been done is this, that if we open the trachea, and air enters below the vocal cords, we put the trachea at rest. After tracheotomy the laryngeal movements of respiration diminish, or perhaps cease entirely, and, moreover, the strain which was thrown on the larynx by the passage of air through the constricted region is removed. Finding that the symptoms are getting steadily worse, it is considered safer to open the trachea, to put the larynx at rest, and so to enable the ulceration to heal. If the operation acts in this way, why hesitate? The general experience in such cases has been this, that if the operation is done sufficiently early, the syphilitic process subsides, and the ulceration heals, but in healing more fibroid tissue is formed, and the physiological use of the passage having been discontinued, it tends more than ever to contract. Before doing the operation we must consider these questions. Will the patient ever be able to live without a tube in his trachea, and, if so, when will it be possible to remove it? In most cases I should say, do the operation if the symptoms are serious, but do not leave the tube in longer than is necessary for the healing of the ulcer. Some patients have gone on wearing the tube for years, for the rest of life. I had a patient once who had his trachea opened for laryngeal syphilis; the ulceration healed, but the larynx had contracted so much that we were afraid to leave out the tube, and he used to come up occasionally for inspection. This went on for a year and a half. The man was a cabman, and it was very important for him to get rid of his tube, so at last I determined to take the risk of leaving it out. We took out the tube and he breathed through the wound; gradually the opening closed, and the power of breathing through the larynx returned. Is the larynx ever affected in secondary syphilis? Yes, it is subject to catarrhal inflammation, but this is transient and never serious.

Catarrhal inflammation of the larynx causing symptoms like those of croup is common in children; it is sometimes known as spasmodic croup, but that name being also applied to another condition which I shall describe presently, I shall speak of this as catarrhal croup. The disease begins like an ordinary cold in the head. In the night the child wakes up with symptoms of croup, with crowing breathing. The doctor is sent for, and the first thing he has to ask himself is, whether it is a case of catarrhal croup or of membranous croup. As far as the crowing laryngeal sound is concerned, it is indistinguishable from that which occurs in membranous croup. If he looks at the throat he sees a congested catarrhal mucous membrane; but he need not look at the throat to decide. In these cases there is never evidence of much obstruction to the entrance of air. Look at the pit of the stomach and you will see that there is no sign of excessive action of the diaphragm. There is much noise, but little or no sign of mechanical obstruction. The temperature is not above 100°, whilst it is usually well above this in membranous croup, and we can tell the parents at once that there is no danger. The usual experience in these cases is that we give the patient an emetic, and in an hour or less the attack is over. These attacks are liable to recur in the same child, and we often find that several children in a family suffer from them, so that we are led to think that there is some family tendency, possibly a disorder of the nervous system. I have never known a child die of this catarrhal croup.

Spasmodic croup occurs in connection with rickets, the attacks being commonly called laryngismus stridulus. I have already described the symptoms of this condition in my lecture on Rickets. Here I will merely say that if we are called to a case of croup and the child is under two years of age we should suspect rickets, and look for signs of that disease. These attacks sometimes lead to sudden death.

I now come to membranous croup, thatis to say laryngitis in which false membrane is formed, so that there is much obstruction to the breathing, and a crowing sound on inspiration. Do not let your minds get entangled by trying to think out the differences between diphtheria and membranous croup, we do not know enough to speak confidently about this matter. Some think that membranous croup is always laryngeal diphtheria. Some years ago a committee was appointed by the Pathological Society to decide the question, but they were unable to come to a decision. I am sure that anatomical appearances do not enable us to answer the question, for a scald of the glottis gives rise to anatomical appearances indistinguishable from those of laryngeal diphtheria and membranous croup. In all cases alike there is the so-called plastic inflammation of the larynx—that is all we are able to say in the post-mortem room.

This form of croup comes on like a feverish cold, the temperature being 101°, 102° or more. Then we hear a croupy cough and crowing breathing, and soon we notice that during inspiration the epigastric region is sucked in, telling us that the air has much difficulty in entering the lungs. Let me repeat that in catarrhal croup we never see this sign. As in diphtheria which has extended to the larynx, but still more in these cases of membranous croup, they are liable to sudden spasm or paralysis of the vocal cords, and they suddenly become livid, pulseless, and die in a minute or two. More commonly the inflammation extends down the bronchial tubes, and they die of bronchitis or broncho-pneumonia. If you are asked the grounds for separating membranous croup from diphtheria, say that the only clear ground for distinction is that in laryngeal diphtheria with other unmistakable signs of diphtheria there is more evidence of contagion ; several persons in the same house may be attacked, while we do not see this with membranous croup. The answer made to that argument by those who hold the identity of the two diseases is that the cases of membranous croup are sporadic cases of diphtheria. I know of no way of deciding the question. One thing however we have to recognise, and that is, that in some families there seems to be a curious tendency to these plastic inflammations. In the course of years one child after another is attacked, leading us to think that there must be an inherited constitutional tendency.

Œdema of the glottis is common in a slight degree in venous congestion, but this is never fatal. It also may occur in connection with erysipelas, and phlegmonous inflammation. Scalds of the larynx, as from an attempt to drink out of the spout of a kettle, may rapidly cause extreme œdema, and the swallowing of poisonous irritants, such as sulphuric acid, will also cause it. Fatal œdema of the glottis is apt to come on in Bright's disease. Another common cause is perichondritis, which I am about to describe. With the laryngoscope we see that there is extreme swelling of the mucous membrane around the entrance to the larynx, so that we can with difficulty see the vocal cords. The swollen epiglottis can also be felt with the finger, or even seen on merely depressing the tongue with a spatula. The mucous membrane has a bright glistening appearance, and when we prick it serum oozes out.

Perichondritis is an inflammation around the thyroid and other cartilages of the larynx, which may prove fatal. On post-mortem examination we find much swelling in the upper part of the throat. We cut into the larynx, and we are struck by the œdema, which is so extreme that we recognise in it the immediate cause of death. We cut into the swelled membrane, and we find pus lying round the cartilages. There is a suppurative inflammation around the cartilages, and we want to know the cause. On cutting into the cartilage we experience a grating sensation, and we find that the perichondrium has been separated from it. There has been necrosis of the cartilage, with some calcification. There is a slow necrosis, and then something sets up suppurative inflammation, and this leads to œdema, which kills the patient. The severity of the disease will depend on the extent to which the cartilage has undergone necrosis. Perichondritis may arise in connection with any of the chronic affections of the larynx—phthisis, carcinoma, syphilis, and simple chronic laryngitis. It also occurs as a sequel to the laryngeal inflammation not uncommon in typhoid fever, and may be due to a blow on or wound of the larynx. If death does not occur from œdema, nor from one of the complications I am about to mention, the cartilage may be exfoliated, and contraction of the laryngeal passage, like that due to syphilis, is apt to come on. Moreover, as the primary condition in connection with which perichondritis has arisen is very commonly itself fatal, recovery from perichondritis is exceedingly rare. Besides the usual symptoms of chronic laryngitis, there is commonly much pain in the larynx, especially on swallowing, and we may detect the formation of an abscess, either externally, or with the laryngoscope.

There is always danger that persistent inflammation of the larynx may excite inflammation travelling down the tubes, and causing broncho-pneumonia, which may go on to an abscess. I remember in a post-mortem examination finding abscesses scattered immediately under the pleura. Like those of pyæmia, they were mostly in the bases of the lungs; but I was not satisfied that they were pyæmic, for instead of hæmorrhagic masses, the pus was contained in areas of red and grey hepatisation. On opening the larynx I found signs of persistent inflammation.

If you ever have to deal with a case of cut-throat, remember the danger of broncho-pneumonia when the throat seems to be healing, and this danger is found in all persistent inflammations of the larynx.

If you are asked, "What diseases in the chest may persistent and severe inflammation of the larynx bring about?" say:

1. Œdema and congestion of the lungs, which may prove fatal.

2. Bronchitis, more or less suppurative, but commonly fatal capillary bronchitis.

3. Broncho-pneumonia, in various stages, down to suppuration.

4. Broncho-pleuro-pneumonia.

If the trachea has been opened and the surrounding connective tissue irritated, and suppuration excited, the suppurative inflammation may travel to the pericardium and kill the patient. This is one of the dangers of tracheotomy—suppurative pleurisy arises in a similar manner.

LECTURE XXIV.

PHTHISIS.

WHAT do we mean by phthisis? The old meaning was wasting away; hence it was termed consumption—that is, a loss of substance and a loss of strength; and that is what phthisis means. The best way of regarding phthisis is to recognise that there are external and internal morbid conditions taking away the health and substance of the body. Phthisis is a rascally robbery of the vilest kind; it is an insidious stealing away. This taking away was for years seen to be associated with coughing up, with profuse sweating, with the passage of excess of fluid substance from the bowels. Phthisis was for centuries recognised symptomatically before it was known that there was disorganisation of the lungs. Later it became known that in phthisis there were holes and cavities and ulceration in the lungs; then that these cavities and ulcerations were associated with little lumps in the lungs, which they called nodes; and still later it was recognised that these nodes were associated with still smaller masses, which received the name of tubercles. Further, it was noticed that with these changes in the lungs and with this wasting away, there were feverish outbursts, and that the expectoration increased and got more sticky in these outbursts, like the expectoration of pneumonia, and after death they found morbid changes in the lung-substance very similar to those seen in pneumonia. Hence it was said that the destroying agency in the lungs was pneumonia, and that pneumonia was the cause of phthisis. But those who had noticed more particularly the tubercles said the destructive agent was not pneumonia, but tubercle. These two contending views divided pathological opinion, and continue to do so.

This regarding phthisis as due to lung destruction is a useless view, not tending to cure, nor sufficiently to prevention. Further observation has revealed finer morbid changes leading to tubercular inflammatory destruction of the lung, and that these finer morbid changes of nutrition in general, and in special structures, are the essential conditions producing phthisis. It was necessary to recognise this fact, for we had to recognise phthisis in a curable stage. Some doctors who had devoted much time to auscultation were guided mainly by the pulmonary signs; others, men in general practice, who saw phthisis from an early stage, saw it attack one member of a family after another, and they looked upon the general symptoms as the best guides. Never go to an asylum if you want to see early stages of insanity, and do not go to chest hospitals to study the early changes of phthisis.

This is the growing impression, that the lung destruction is the final effect. A poor woman was dying, wasting away and losing strength. The friends asked, "Is it not consumption, doctor?" "Yes." "How is it, then, that she does not expectorate?" Only at the last was a little lung disease detected. There are some cases of phthisis in which a surprisingly small portion of lung is affected. It was looking for finer morbid changes that brought forth the bacillus investigation and revealed those feeders on the dead.

In the early periods of phthisis there are various special disorders and various general disturbances, showing a failure of physiological functions. These have been collectively termed the pre-tubercular changes. One condition that has been noticed is that there is a build of body which disposes to phthisis: a long, narrow and shallow chest; with this narrowness an extreme obliquity of the ribs, which are much depressed; with this also flattening under the clavicles, and an evident tendency to sinking in of the supra-clavicular spaces; all these things pointing to diminished capacity of the breathing organs—in other words, to weakness in these organs. That form of chest is usually associated with slender bones, with narrow and weak muscles, with an excessive sensibility in nerve-tissue, the veins stand out unduly, the features are usually finely cut. I told you of the relation between struma and phthisis, but the strumous constitution is not the only one which leads to phthisis. Some have wrongly called these people strumous, but they are quite different from those of the true strumous type, who have thick lips and thick alæ nasi. The narrow-chested, nervous persons seem born to become phthisical, and they are the gloomiest cases of phthisis, the cases of tubercular phthisis in which, as Addison said, there is no sign of repair.

The excessive sensibility in nerve-tissue noticed when phthisis is tending to come on affords much quickness in feeling, but also much restlessness. These persons are characterised by increasing restlessness which tends to wear them away, leading to much mal-nutrition. What do we mean by restfulness? We mean certainty. Therefore, restfulness is always an indomitably strong condition, a condition in which we are able to circulate to the fullest extent. The excessive sensibility and quickness of feeling lead to many troubles in the sphere of consciousness; therefore these constitutions are much troubled with imagination, and in dealing with the world's actions they usually get "over-strained," exceedingly anxious, and this tends to cause tightness in breathing and weakness in blood circulation. Their restlessness deprives the body of energy and heat, and they have usually cold extremities; and this tightness in breathing early affects the venous circulation, which leads to the standing out of the veins of which I spoke before. Owing to the weariness and exhaustion the mind is much disquieted and much bewildered. There is that thought which comes up always when the human feeling is not comfortable and resting, "What does it mean?" With that comes alarm and timidity; and one of the most important things we have to do in phthisis is to keep patients out of this dread and confusion, because it causes a terrible depression, and the blood commonly gets whiter and whiter.

The patients complain of being "nervous," while their friends say they are "excitable." They are in a condition in which they cannot healthily correspond. Then the feeling becomes very depressed; appetite is feeling demanding; hence their appetites become very depressed. Not only the appetite for food, but for other things as well. They have a tendency to stay indoors too much, they do not care to go to mother earth; they do not care for meat and vegetables, but for messes.

Some of the nicest people we come across are those who have the phthisical tendency, but they are some of the saddest people; melancholia and tendency to phthisis go together.

Following these remarks, it becomes clear that a large proportion of phthisis is inherited, but that in the inheritance there are conditions which are removable if healthy surroundings are established. Time has shown that many of these people become strong and healthy. We must learn to look out for the earliest manifestations of the phthisical tendency. The children in phthisical families often waste away with weak digestion and recurring pneumonia. Or they may grow up, and at about the time of puberty we may see signs of failing nutrition. They have loose flesh; they are anæmic and subject to catarrhs; we often wonder if they will die of pneumonia or other lung disease.

You will learn from patients with the phthisical tendency that, owing to their excessive sensibility, they feel changes in the weather very much, and are usually very liable to catch cold. They frequently have a difficulty in deciding how to dress suitably. This question of sensibility comes up very curiously, and with it the most important question, "Do you think he is properly clothed?" The object in clothing is to provide an artificial skin. Feathers and wool are beautiful instances, they imprison a layer of still air, and so keep the body warm. The test of clothing should be that the mind is neither harassed by cold nor worried by heat.

It has further been noticed that with this tendency to catch cold, nutritive disturbances in various mucous membranes occur; catarrh of the stomach, catarrh of the windpipe and throat, and catarrhal diarrhœa are common, so that recurring diarrhœa is itself a warning of phthisis. Repeated indigestion, with pain after food, with nausea or actual vomiting, bringing up of mucus, with loss of flesh, may warn us that there is some constitutional weakness, and we should look out for phthisis. There is liability to ostitis passing on to caries, liability to eye inflammations. Catarrh of the skin is also common, and catarrhal inflammation of the middle ear. Palpitation is another symptom, and in some cases of early phthisis is the symptom of which patients complain most, and which brings them to the doctor. With this palpitation and restlessness we have commonly to deal with disturbed nights and excessive dreaming, and usually with this disturbance of the mind in sleep there is much mental depression.

Phthisis commonly comes on with anæmia, recurring, increasing, irresistible anæmia. In elderly people especially phthisis is apt to be very obscure. For a long time the only signs may be anæmia and weakness. Anæmia increases and increases throughout phthisis. The blood is wasting away, and not only the red corpuscles, even the water sweats away—the night sweats have long been counted amongst the most characteristic symptoms of phthisis. As the red corpuscles, the great breathing organs of the blood, waste, muscle loses its activity and the heart becomes weaker and weaker. I told you that death by anæmia is death from fatty degeneration of the heart. This is why palpitation is so common in phthisis. With weakened heart irregular circulation in the brain comes on, and this leads to distress in the head; they are confused and wearied, and cannot bring their minds to any decision. In other cases there is weakness not only in the heart, but also in the vessels; epistaxis comes on, and may be profuse. The skin is, of course, pale, owing to the anæmia, but it often has a yellowish tinge, and it may be much pigmented. Some of the most pigmented skins I have seen have been in phthisis. If asked, "In what conditions do we meet with extreme pigmentation of the skin?" say, "Phthisis, Addison's disease, and malaria."

With the failure of muscular and nervous tissue, we observe failure of another transmitter of energy, elastic tissue. This comes on most markedly in the lung, especially at the apex, though it may be all over the lung; this failure of elasticity usually affords the earliest physical sign of coming phthisis. There is loss of elasticity in the upper part of the lung; if phthisical change is spreading, the loss of elasticity extends into the middle of the lung; if phthisis is tending to kill, this loss reaches down to the bases of the lungs. What are the early signs which precede consolidation, and which indicate loss of elasticity? Upon the elastic tissue depends the vesicular action of the lung, and this action gives rise to the sound known as vesicular breathing. With the atrophy of the elastic tissue the vesicular breath-sound is lost, and is replaced by a harsh tubular sound, whilst, again owing to the deficient elasticity, the expiratory sound is unduly prolonged.

Sometimes the vesicular breathing is lost to such an extent that we hear very little; the breath-sounds are said to be muffled. With this there may be disturbance in the mucous membrane of the bronchial tubes, attended by clicking sounds, or, as it used to be called, a "cog-wheel sound," and often bronchial râles supervene. Whenever a function is failing, the structure wastes; therefore atrophy comes on as the lung function fails, and the percussion note becomes flat; there is diminished resonance, with a wooden note—then we do not say that there is consolidation, but that there is less air in the lung. This atrophy is usually more marked at one apex than at the other. Remember that phthisis never attacks both apices equally, but generally one before the other, or one more than the other. There is an atrophy of the apices which comes on emphysema, and there is a prolonged expiratory sound, denoting that the elasticity of the lung has failed, but this failure in emphysema is about equal on the two sides. As we look at the chest we see external signs of the changes that are going on within; there is flattening of the infra-clavicular spaces, sinking in of the supra-clavicular spaces, and on looking at the back we notice that the scapulæ are unduly prominent. If the chest is thus wasting away, with failing circulation and increasing depression of feeling, never be surprised by the onset of phthisical destruction of the lung. I have seen it come on in almost any disease, where there was that condition of the chest.

In this stage the disease is remediable. I will even say that it is curable, as far as such a constitution can be cured. I mean that they can be guided to become much stronger. But can we say that there is no tubercle in the lung? No. But can we hold out hope? Yes. On the other hand, I have known them die rapidly when tubercle was not suspected, and its presence has only been revealed on post-mortem examination. The disease has come on insidiously. Why has it been so insidious and so rapidly fatal? My conclusion has been that the circulation through the lung became weaker and weaker, and the plasma, the endothelium of the capillary walls, the lymph elements in the peri-vascular spaces, the circulation of all became weaker and weaker, and albuminous matter accumulated and was incapable of active development. Owing to the weakness of circulation there was a low form of nutrition, and a rapid degeneration of tissue took place.

There is commonly another symptom, and this is feverishness. If there be feverishness without local signs of disease to account for it, the temperature rising at night and falling in the morning, a difficult question may arise as to whether there is any lung disease. There may be a small patch of pneumonia, which we may fail to detect by physical signs, and yet it may cause much fever. It is notorious that early phthisis may be attended by much wasting and loss of strength, and may go on for months before we can pronounce the lung diseased, and this uncertainty may produce in the patient a feeling of despair. "I am in heaven," were the words of a young woman who before death lost this fearful feeling.

Usually, however, within a few weeks, or it may be within a few days, of the onset of fever, we notice increased dulness, some bronchial râles, and increased voice-sounds, signs of consolidation; we also observe that expectoration and cough have become marked symptoms. The muco-purulent exudation denotes catarrhal inflammation, whilst if it is very viscid, sticking firmly to the vessel, it points to pneumonia; we may often gather the nature of such acute inflammation from the expectoration.

It should always be recollected that acute pneumonia may attack the apex of the lung in a patient much disposed to phthisis, and yet clear up, and the patient entirely recover. Do not overlook that fact. Perhaps a young woman has died of phthisis, and another sister is attacked in this way by apical pneumonia, and the parents of course dread consumption. In these cases the symptoms are those of acute pneumonia, the consolidation has come on rapidly with feverishness and cough, there is dulness, bronchial breathing, and bronchophony.

Usually in the earlier course of phthisis the general symptoms subside, but signs of consolidation are left behind; the patients recover their strength and appetite. After a time there is a recurrence, another feverish outburst, and more signs of consolidation, and therewith we may notice much crepitation. Is the lung disorganised, is there a cavity? This is not easy to determine in the early stage, because the large crepitation may be due to exudation in the larger bronchi, which later may entirely disappear; but, on the other hand, we cannot say positively that there is no cavity, even in the entire absence of the signs of cavity, because there may be very small cavities away from the surface that we cannot detect. After a while, however, we notice with large crepitations undoubted signs of cavity. There is cavernous breathing, and we may hear the hollow ring of coughing in the cavity. If we ask the patient to speak we hear the sounds of the words intensified, reflected from the walls of the cavity—this is what has been called pectoriloquy; the crepitation is usually very large, a gurgling sound, so that if we ask the patient to cough we hear the gurgling and the cavernous sounds together. What conditions must be present to enable us to determine that there is a cavity? The cavity must have a free communication with a bronchial tube, to let air pass to and fro in the exudation, and to conduct the breath and voice sounds to the cavity; and it must be situated, if small, near the surface of the lung. If the cavity be small and in the middle of the lung, or if the bronchial opening be closed, it may entirely escape notice.

In the course of phthisis we are never surprised to find that pleurisy comes on, and that at any stage of phthisis; again, whenever we find signs of pleurisy, we always examine for phthisis, our reason for doing this being that in some cases pleurisy is the first indication of phthisis. If we find a patient attacked with acute pleurisy, and learn that he has been losing flesh and strength, then we are apprehensive of phthisis. In such cases we may only find flatness on percussion at one apex, and hear harsh breathing and prolonged expiration, but from the fact of pleurisy, coupled with the loss of flesh and strength, slight as these signs are, we should take precautions against phthisis. If the phthisical change in the lung be breaking down, with suppuration, and is close to the pleura, the pleurisy commonly passes into empyema, or suppurative inflammation of the pleural cavity. Phthisis is the most common antecedent of empyema, so that in all cases of empyema we examine carefully for phthisis. Once upon a time in treating pleurisy we used to direct our efforts to stopping the inflammation of the pleura. Now suppose we could stop the pleurisy (we cannot), when there is suppuration close to the pleura, what would happen? The two sides of the pleura would not become adherent. What would happen then? Pus from the cavity would perforate the pleura, and escape into the pleural cavity, and would give rise to empyema, and very likely air would come too, causing pneumothorax. Therefore we seek to guide the pleural inflammation safely, so as to prevent empyema and pneumothorax; for the best thing that can happen is that the two sides of the pleura should become adherent. If adhesion occurs, pus cannot accumulate, nor is there any risk of pneumothorax.

Other intercurrent inflammation is common with phthisis. Nephritis is liable to come on, leading to large white kidney, and this may arise very insidiously. Another associated inflammation is perityphlitis, and in some persons such cæcal inflammation may precede the onset of phthisis. Never fail to examine the lungs in cases of "fistula in ano," for this condition is commonly associated with phthisis. There may be catarrhal inflammation of the bowel leading to ulceration, with or without tubercular deposit, and this inflammation may set up protracted diarrhœa. Catarrhal inflammation of the windpipe causes most distressing cough. The quacks with their "inhalers" do a large trade with these patients. Catarrhal inflammation of the larynx may pass into actual phthisical destruction. This is the most distressing form of phthisis. In some cases the irritation is so bad that you cannot get the patient to take food. Acute changes also occur in connection with the brain. They may take the form of tubercular meningitis, or of epileptic attacks, or of melancholia. There is a peculiar relation between phthisis and insanity. We find cases of melancholia alternating with attacks of hæmoptysis. When the lung disease is most active the melancholia passes away, and *vice versâ*. At post-mortem examinations in asylums phthisical destruction of the lungs is often found, and this may be in cases in which the existence of phthisis was never suspected during life, the disease having been entirely latent. Therefore in all cases of insanity, especially in young subjects, examine for phthisis. Acute mania and melancholia are the forms of insanity most commonly seen in connection with phthisis. Do not, because there are fits, assume that there is tubercular meningitis; they may be due merely to failing nutrition of the brain. There may be less severe nervous disturbance, taking the form of hysteria, the feeling becoming restless and ungovernable as they pass into phthisis.

In the course of phthisis we are often impressed by the increasing anæmia, and it would be a fair question to put, "What is the relation of anæmia to phthisis?" First, members of families disposed to phthisis are very subject to anæmia. In such persons the anæmic tendency is a constitutional feature, recurring anæmia with more or less wasting. Secondly, anæmia may persist with wasting and resist all treatment, and during that time phthisis may be insidiously creeping on, so that for weeks or months it is a disputed point as to whether there is or is not lung disease; hence whenever there is anæmia and loss of flesh, with flatness on percussion and harsh breathing under the clavicles, we should always consider that the anæmia may have some relation to threatening phthisis. A mother brought her daughter with anæmia, and asked what made her so exceedingly weak. The symptoms had evidently resisted all treatment. I could not discover any organic disease, but there was prolonged expiration and harsh breathing at one apex, with loss of flesh. I could not be certain that there was phthisis. The patient was admitted to the hospital a few months later, where she died with acute pleurisy and acute phthisis. Again, a young woman was in hospital with anæmia; her temperature rose a little in the evening, and there was a prolonged expiratory sound heard on listening under the clavicles. The patient was under observation for several weeks before phthisis could be diagnosed with certainty.

These are the gloomiest cases of phthisis, because where there is anæmia the disease usually is rapidly progressing. With anæmia we always notice weakness of the pulse. What is the worst feature in phthisis? I should say, weakness of the general circulation. How do phthisical patients die? The circulation gets weaker and weaker. When measuring the severity of phthisis do not be guided by the signs in the lung only. Many die with exceedingly little lung mischief. On the other hand, I have seen cases which lasted for years with extensive destruction of the lung. I remember especially one case in which we found at the post-mortem examination that the whole of one lung was practically a large cavity. A rapid, exceedingly soft pulse is one of the gloomiest signs in phthisis, especially where there is much anæmia.

Year by year I am more and more impressed by the fact that they hardly ever die of suffocation. At the post-mortem examination we find the left ventricle flaccid and more or less full of blood, whereas in death from suffocation, as in drowning, in strangling, and in laryngeal diphtheria, we uniformly find the left ventricle contracted and empty, while the right ventricle is distended, showing that the blood could not get through the lungs. They die in phthisis from loss of circulatory power in the heart and great vessels. Why do these cases with huge cavities in the lungs last so long? Because there is power in the blood. It is marvellous with how little lung creatures can live, provided only that the blood can flow rapidly enough past the oxidising surface. The whole treatment of phthisis is to watch the signs, to see what is wasting away, and use it as a guide to enable you to give the patient a stronger circulation. How are we to do that? Remember the value of hope and do not depress the patient. Spenser well describes the paralysing effect of fear on the circulation, "He stood pale and blue, and sweated profusely."

Three symptoms appear early in these cases of phthisis with anæmia and weak general circulation—very feeble pulse, cold extremities, and tendency to venous congestion and œdema. With these conditions there is usually another symptom—loss of healthy feeling and inclination, which is generally manifested by loss of appetite. You will find, when you consider people complaining of loss of appetite, that you cannot regard it merely as mouth or stomach failure, but that often it is a manifestation of mental depression ; therefore sometimes a little firmness on our part will do more than medicine. These patients want clearness and certainty given to their minds as to the way they ought to go. Encourage hopefulness in a patient, and a healthier feeling usually comes. With the loss of appetite, which is one of the most frequent symptoms in phthisis, patients become weaker, until sometimes they cannot even take food. You may have to use the stomach tube if the patient refuses all food. When food is introduced the gastric and other functions become more healthy. What is the natural means to get stronger breathing ? Food ! What is the natural means to get stronger circulation ? Food ! If necessary therefore do not hesitate to pass a tube down the gullet, and so to feed the patient.

In the course of phthisis we are never surprised to find blood in the expectorated matter. Hæmoptysis in some degree is met with in about 80 per cent. of all cases of phthisis, but it is only the cause of death in about 5 per cent. Have these facts firmly fixed in your minds. When I was physician at Victoria Park, I asked Mr. Power, the resident medical officer, to examine the records for many years, and to ascertain the proportion of fatal to other cases of hæmoptysis. The result was that given above. In Sweden a similar examination was made, with a similar result. By far the majority of people who have hæmoptysis in phthisis do not die of hæmoptysis, and we can therefore relieve their minds immediately by saying that they need not be apprehensive. What are the features in hæmoptysis which will lead us to be apprehensive of a rapid death ? When bleeding from the lungs kills, it is by a great rush of blood filling up the bronchial tubes, shutting out the air before the patient can take a deep inspiration in order to cough the blood up. How is this known ? From post-mortem examination. Some patients cough up blood, suddenly die, and the bronchial tubes are found choked up with blood clot. When a patient is bringing up large quantities of blood very rapidly, perhaps half a pint of blood or more in a very few minutes, we cannot but ask ourselves, "Will this patient, with the next gush, be suffocated before he can get the blood up?" I have only seen one case of death from hæmoptysis in the early stage of phthisis. Almost always fatal hæmoptysis is in old cases of phthisis, with cavities, and more or less fibroid repair. A vessel lying in the wall of a cavity may have very little support, so that it bulges into the cavity, becomes aneurysmal, and bursts. I will give you a striking instance of this condition. A man was admitted to the Victoria Park Hospital bringing up large quantities of blood rapidly. He had not phthisis, but he had a cavity in the lower part of his right lung, which had been produced by fracture of a rib, and this old cavity I found choked with blood, and a ruptured aneurysm in its wall. This man was known to the resident medical officer, who had seen him at the time of his accident, years before.

It is a terrible thing for a poor patient to be coughing up blood. One of the bravest men I knew, a captain in the army, had profuse hæmoptysis, and I had to quiet his fears. I was anxious to get him home alive, and succeeded in doing so. I helped him by saying "Cough it up, captain!" Fear tends to paralyse the breathing and to paralyse the right side of the heart. What chance has a poor fellow if he is coughing up blood and is taken with fear? To another man with livid face and with a fearful pallor, I said "Cough it up all you can, for it will help you."

Moderate hæmoptysis often occurs several times in phthisis, and such cases are often of long duration. This leads us to wonder to what extent hæmoptysis is unfavourable. It is not necessarily unfavourable. May it not be that bleeding in some cases relieves congestion, and prevents rapid death by pneumonia? I have no doubt that in some cases hæmoptysis arises from, and relieves, the congestion which precedes pneumonia. In these cases the flow of blood is commonly preceded by feverishness, and may be accompanied by other signs of inflammation. Hæmoptysis comes on sometimes in an astonishing manner in persons who are not suspected to have anything wrong with the lungs, but soon afterwards, unmistakable signs of phthisis supervene. This has led some medical men to speak of such cases of phthisis as caused by hæmoptysis. Some weakening condition, they consider, leads to hæmorrhage, and this sets up a "low" form of pneumonia, which ends in phthisis.

In considering phthisis and the treatment of phthisis, do not think of the lungs so much, for it is simply harassing to one's self, and leads to the death of the patient, and no one benefits by it. How can a lung be repaired if there is not sufficient blood going through it? The one object in the treatment of phthisis is to get more blood through the lung to repair it; hence the importance of rest, food, and fresh air: poor patients benefit so much by coming into the hospital. If people are shut up indoors too much, their circulation becomes weak; if outdoors sufficiently, their breathing is freshened and their circulation strengthened. All experience proves that phthisis comes from excessive indoor life, from abuse of domestication. This applies to plants with too much forcing and heating; they waste away, as well as animals and men. The cows kept in London die of phthisis, and so do the horses. I have often had phthisical lungs brought to me from the knacker's. Horses do not get inflammation of the lungs when they are out in the fields in all weathers; it is when they are stabled. The animals in the Zoological Gardens die of phthisis, especially the monkeys, when they won't let them be monkeyish. Hence we see phthisis mostly among persons who are too much indoors—nuns, sisters of mercy, bakers, tailors, law-writers—in fact, wherever there is too much domestication. If we take bellows and blow into each others faces, we shall draw deep breaths. There is the same effect produced by a dash of cold water and a dash of cold air, they free the breathing. Of course, if there is not enough movement in the air, if the air is too much confined, the breathing must get weaker and weaker, the chest movements shallow, the ribs oblique, the chest flattened, and atrophy of the lungs follows. I first learned to understand, from reading Darwin's "Animals and Plants under Domestication," how diminished use leads to atrophy.

Whoever was associated with a lot of people indoors without being made anxious and restless? When indoors too much we become exclusive, we know everything. Ancient Egypt shut out the world at large, and rotted out from care and ignorance, with vitiated air, liability to cold, and phthisis. Phthisis is said to be due to bacillus, but of what use is that view in curing phthisis? Do not be content to love knowledge for the sake of knowledge, but for what you can do with it. The view is inadequate to cure phthisis. We have to look upon phthisis as made up of many physiological disturbances, and it can only be cured by bringing these disordered physiological changes into order again; and in this way hope in the cure of phthisis can be entertained. Are there any changes in the lung that will warrant us in thinking that phthisical changes can be cured? That question brings us to consider next the morbid changes in the lungs.

Cases of phthisis have been grouped according to the character of the morbid changes in the lungs. It has long been recognised that in a large number of cases there are so-called tubercular changes, while in other cases we find pneumonic consolidations which have disintegrated and destroyed the lung, but few or no tubercular changes. These last have been called pneumonic phthisis. Those in which the lung is studded with groups of tubercles, with comparatively little pneumonia, are known as tubercular phthisis. In the majority of cases there is a mixture of tubercular and of pneumonic changes, and these are called tubercular pneumonic phthisis. That is a clear natural grouping.

In the pneumonic cases the symptoms come on much like those of pneumonia, which persists and spreads and cannot be arrested; this is the so-called galloping consumption. The pneumonia begins at one apex, spreads down towards the base, and attacks the other lung; the temperature, expectoration, and cough, are very like what we see in ordinary acute pneumonia, but the lung disintegrates. In other cases it begins in the form of small patches of broncho-pneumonia. These cases are, for a time, exceedingly difficult to diagnose. During the first few weeks or months there is recurring cough and expectoration, with high temperature and loss of flesh and strength, and sooner or later undoubted physical signs of phthisis appear. In making the post-mortem examination in such a case we are struck by the appearance of a number of small masses, the remains of pneumonia. At the apex there is grey hepatisation which has passed more or less into a caseous condition. As we approach the base of the lung there is less and less caseous matter. There we see small masses of red and grey hepatisation of about the size of a threepenny piece. In the middle of the lung these masses are evidently confluent, increased in number, and pressed together to form large aggregated masses. At the apex the pressure has obscured the outline of these masses, so that we can no longer make out their aggregated structure, and in the midst of this material we see a number of cavities, many with a kind of lining membrane, showing their long persistence. Both lungs are similarly affected, but one to a more extreme degree than the other.

In the tubercular pneumonic type, of which the majority of cases of phthisis are examples, the disease advances in a series of attacks, with arrest and improvement in the intervals, as I described in an earlier part of this lecture. If we examine a piece of lung in which there has been broncho-pneumonia and subsequent caseation, we find the air-cells filled with granular matter, consisting of fat granules, broken-down corpuscles, and more or less pigment, and in the interstitial tissue of the lung we find a similar material. Is this tubercle? The question is still open. Laennec called it "mature tubercle." In tubercular pneumonic phthisis, on examining the base of the lung post-mortem—to see the most recent formation always look at the base—we see a large number of masses about the size of large shot, of a bluish-grey colour, and more or less opaque. Addison called these "boiled-rice tubercle." They are aggregated together, very friable, semi-translucent, granular, and differ from ordinary miliary tubercle. At the base we find a large number of them, distributed in groups of twos and threes; but as we pass up towards the middle of the lung, the groups get larger and larger, until we find masses no longer tubercular, an inch in diameter, but of tubercular origin, and with tubercles scattered round them. These masses increase in size and are more and more welded together as we approach the apex. As well as the tubercular change, there is broncho-pneumonia: the masses I have described are surrounded by red and grey hepatisation. On close examination we see many degrees of translucency, but the masses are mostly opaque and yellowish in the centre. As the masses get welded together the caseous cores get larger and larger, and finally break down in the centre, forming cavities.

Now that we have come to regard phthisis as a recoverable condition, it becomes important to know what are the actual destructive changes which break up the lung and lead to the formation of cavity. Laennec said it was caseous softening in the centre of tubercle. Addison said that Laennec was wrong, and that excavation was the result of broncho-pneumonia. Both views had a basis of fact, both were right as far as they went, but were too narrow. Look at the lung, see the cavity with the remains of inflammatory deposit accumulated round it, consolidating much of the upper lobe. Of what do these remains consist? Largely of yellow dead caseous material. This is a fine granular material which obscures the light; it is dark and formless, confusion confounded, so that it evidently can take on no organisation, but is quite useless. It contains a large quantity of pigment derived from broken up red corpuscles; it fills up the air-cells and presses on the capillaries. When we see it we say, "That is the remains of inflammatory deposit and tubercle; it has led to the formation of a cavity by pressing on the vessels and causing them to become plugged." How do we know that the vessels are plugged? We see them plugged on post-mortem examination, and we know that such plugging of vessels must give rise to necrosis. It is this thrombosis which makes death from hæmoptysis comparatively rare. What else destroys the lung? As the capillaries are pressed upon by the tubercular deposit, the blood is hindered, and the vessels nearer the heart become over-distended. The blood escapes from them and sets up pneumonia, which also leads to destruction of the lung. Laennec and Addison were both right; both pneumonia and tubercular necrosis break up the lung.

In these lungs it is not uncommon to find the signs of repair. What are the signs of repair? Scar-like material, such as we find where an ulcer has healed, or where a hydatid has been buried up and rendered harmless. We speak of it as the fibroid material of repair. These are the changes in the lungs when phthisis has been arrested and the patient has been comparatively well for years, and they warrant us in saying that phthisis may be arrested and the lung repaired.

Tubercular phthisis must be carefully distinguished from disseminated miliary tubercle in the lungs. Some have doubted the existence of this condition, which is rare. In this the "boiled-rice tubercle" is present in enormous quantities—it increases and increases and forms ever larger masses, until the upper lobes are one mass of consolidation. In parts it is so homogeneous that some have called it tubercular infiltration. We see a semi-translucent, grey, finely granular, "vitreous" material. There is comparatively little pneumonia, but much caseous matter, much pigment, and usually several cavities. It was in examining this condition of phthisis that Addison used to say, "No repair." This is the phthisical change that especially occurs when there is the phthisical build of body—in those I spoke of as being born to become phthisical.

We will now pass on to consider the remaining forms of phthisical lungs, namely the fibroid conditions. I told you that in many phthisical lungs we find not only tubercular and pneumonic destruction, but some results of repair. In the course of years of experience we find that some patients are attacked by phthisis, and go from bad to worse, but after a time the disease seems to be arrested, and they may remain much better in health for months, or for two or three years; then they may have another outburst of symptoms, and again recover, and this may be repeated and repeated over years. Such a patient may be attacked by bronchitis. We may notice the signs of bronchitis spreading through the lungs, and our attention is riveted by the excessive breathlessness. The bronchial râles increase daily, and are heard all over the chest, and the action of the right heart is much increased. We conclude that there is acute tuberculosis. With these chronic phthisical conditions patients are liable to acute tuberculosis, and we may find miliary tubercles in nearly every organ in the body. You will remember that the chronic cases are those in which sudden death from hæmoptysis does occur; but, on the other hand, they may have recurring hæmoptysis for years, so that doctors have said, "These cases with hæmoptysis do better." Rapidly fatal acute pneumonia may come on in such cases, and therefore I always look out for old changes in the lung when I have to make a post-mortem examination after acute pneumonia.

In the course of years the physical signs in these chronic cases may become less marked, so that where we heard dulness there may be resonance. In fact, the medical man then in attendance usually puts the case down to emphysema. I have often said, whilst examining the chest and hearing the opinions, "We cannot always diagnose an old phthisis, for the old phthisical deposits in the lungs are buried by emphysema. The air-cells around the consolidation are dilated, and owing to the emphysematous change the lungs are more resonant, the dulness no longer apparent, and even a cavity may be much concealed in this manner." If a patient happen to die with such conditions, you might call it fibroid phthisis. So, when there was a discussion some years ago upon this subject, one party said, "Here is fibroid material and a cavity; I call this fibroid phthisis." But the other replied, "Yes, but when that morbid condition was in an early stage it was ordinary tubercular phthisis, therefore what is the practical use of calling it fibroid phthisis? If terms are worth using they must be true." In one case I saw one lung cirrhosed from end to end, the other studded with tubercle. There is no doubt that there is a large number of cases which begin like ordinary phthisis, but in the course of years pass into a fibroid condition, so that the two conditions merge into one another. Other cases have been noticed in which a patient has been attacked with pleurisy or pneumonia, and in which the physical signs of consolidation supervene or continue, so that we are led to consider that pleurisy or pneumonia has set up a fibroid condition of the lung. I can give you an example of a man who was in the hospital for a time with pleuro-pneumonia, and died many years later, and we found fibroid change in the lung.

There are two other distinct forms of fibroid degeneration of the lungs.

In the first class of cases the patients are usually men who have been strong and well-built, with teeth remaining sound at middle or advanced age, and with well developed muscles—men who have lived hard and drunk hard or had syphilis. They tell you they have had no illness except bronchitis every winter. Stokes calls these cases "phthisis following bronchitis," and so does Andral. I found that men of this type died with cavities in their lungs, and the disease was looked upon as phthisis, but the cavities were little or not at all associated with tubercle. When I looked at the so-called tubercles under the microscope, I found them made up of spherical and spindle cells, which were part of the fibroid material, and it was evident that the "tubercles" were nothing more nor less than young fibroid tissue. In addition to these small bodies there were masses of tough fibroid material from an inch in size up to that of a lobe or more. These patients suffer from emphysema and bronchitis for years, and then they begin to waste, and the cough does not leave them in the summer as it used to. We find continued and increasing dulness in the upper part of one lung. We ask ourselves, "Is it pneumonia?" No, it is not pneumonia, for the temperature is normal and the onset slow; besides the dulness is too great for pneumonia, and when we percuss we feel as if there were something tough, there is so much resistance that we think of fibroid tissue. Slowly the change creeps on from apex to base and attacks the apex of the other lung. Now, these patients do not usually die by asthenia, as in ordinary phthisis, but by capillary bronchitis. They may live for many years, provided the dilatation of the right heart, which is a constant feature, does not become excessive. Where the obstruction in the lung becomes extreme, so that the dilatation of the right heart is very great, lividity comes on, and sooner or later œdema of the feet and ascites. The fibroid change in the lungs may be associated with cirrhosis of the liver, or with granular kidney. Besides the liability to death from capillary bronchitis, these patients are liable to die rapidly from profuse hæmoptysis, from gangrene of the lung, or from acute pneumonia.

In other cases the fibroid material is found as a thready, tough, fibroid network, beginning very commonly in the upper lobes and spreading more or less through the lungs, destroying the air-cells and the bronchial tubes. There is generally more or less emphysema, and there is often general fibroid disease. In such a case the patient has suffered from bronchitis, which has recurred for many years. We percuss, and we find under one or both clavicles dulness and more or less crepitation. There are signs of general bronchitis, and we may think of phthisis with general bronchitis, but there are no signs of cavity. The right heart becomes much dilated, and death occurs from bronchitis.

What is the relation of phthisis to other diseases? Years ago I thought that there were some diseases which were antagonistic to phthisis, but my mind has completely turned round with regard to rheumatic fever, the presence of which was said to exclude phthisis. Struma and rheumatic fever, phthisis and cancer—these diseases have been regarded by family medical men as antagonistic. They said, "We seldom see a patient with well-marked signs of phthisis suffering from rheumatic fever." Hence they inferred that there was an antagonism. Further observation has shown that where there is a tendency to phthisis in a family, one brother or sister may have rheumatic fever, another struma, one phthisis, another epilepsy. There is some curious relation between phthisis, epilepsy, and rheumatic fever. At the Victoria Park Chest Hospital we found from the records that many patients had had rheumatic fever before, but in no instance in which these patients were said to have had rheumatic fever was there any valvular disease of the heart discovered. This and other experience led us to recognise that acute rheumatic gouty conditions, especially in young people, are not uncommonly the precursors of phthisis. I have very rarely seen contracted mitral orifice and phthisis together. Heart disease and phthisis are considered to be antagonistic, but they are certainly found together in some cases.

LECTURE XXV.

VESICULAR EMPHYSEMA.

By vesicular emphysema we understand dilatation of the air-cells of the lungs. The air-cells are excessively inflated, and they usually dilate in groups to form little bladders or large vesicles on the surface of the lung. If they occur along the margin of the lungs only, the condition is termed marginal emphysema, and little or no importance is attached to it. If the air-cells become dilated in the vicinity of masses of consolidation, we speak of the condition as compensatory emphysema. Owing to the choking up of a portion of the lung by solid exudation it is unable to expand, and undue traction is exerted on the neighbouring portions of the lung, so that the air-cells of these become unduly dilated. To this condition also little importance is attached—it is said to be merely accidental.

In some cases we find the groups of dilated air-cells scattered freely over the surface of both lungs. This condition is known as lobar emphysema. The excessive stretching of the air-cells is connected with loss of elasticity, and when we see these groups of dilated cells on the surface we infer that there are probably many lesser dilatations throughout the lung; we think that what we see is indicative of a much more general morbid change, and that there is a loss of elasticity throughout the lungs.

Cases of vesicular emphysema have been arranged by long experience in two groups:—1. Those in which the lungs are smaller than natural. This condition is called atrophic emphysema. 2. Those in which the lungs are much larger than normal. This condition has been called hypertrophic emphysema, but the term is a confusing one, as hypertrophy may be healthy.

In cases in which the lungs are smaller than normal, we observe that the chest is flattened, and that the flatness increases as the case progresses; the infra-clavicular spaces are flat, and the supra-clavicular spaces are sunken. Such emphysema is commonly attended with general wasting, so that it is particularly marked in some old, shrivelled people; and usually when this form of emphysema occurs at middle age it means premature old age, with wasting of muscle, excessive sensibility of body, restlessness, and shrinking from the world—wasting away. With atrophic emphysema, on listening to the lung, we usually hear harsh tubular inspiration and prolonged tubular expiration, there is a flat percussion note over the upper parts of the lungs, and hyper-resonance over the lower.

With the other form of emphysema, we have enlargement of the chest, the barrel-shaped chest described in books, a rounded chest with the supra-clavicular spaces bulging out, and swelling up markedly when the patient coughs; patients have often asked me what that swelling was. It is due to the extreme inflation of the apices of the lungs. The dilated lungs overlap the heart, so that the heart's dulness is diminished, and the sounds are muffled and distant. The enlarged lung also pushes down the diaphragm to some extent, and overlaps the liver, so that the liver dulness is diminished. In this form of emphysema we usually find the percussion note extremely resonant. There are cases of emphysema in which the atrophy and the enlargement are much blended.

Owing to the deficient elasticity of the lungs, and the dilatation of the air-cells, the lungs become excessively distended with air, and the air is not sufficiently moved in and out; it is to a large extent stagnant. As the disease advances we notice that the tubular breathing becomes more and more marked, and the vesicular breathing is lost. In later stages we may be struck by the absence of breath-sounds, the air is so stagnant. And though the breath-sounds are absent, the chest is very resonant. After death in cases of emphysema, when we cut into the lungs, we are struck at first by their extreme paleness, but we soon notice that the cut surface turns scarlet. Thus, by post-mortem examinations, we have been led to conclude that stagnant air was the immediate cause of death. What happens when there is an increasing quantity of residual air in the lungs? The patient is observed to be weaker and weaker, duller and caring less, his power for liking and thinking is diminished, his mind droops, his appetite fails, and his muscular power diminishes.

It has been recognised that when the interchange between the air and the blood is hindered, it becomes difficult for the blood to get through the lungs, and hence the right side of the heart tends to get excessively distended; but there are in emphysema several other conditions hindering the circulation through the lungs, and leading to dilatation of the right heart. If you examine the lung carefully after death from emphysema, you will find that the dilated air-cells have pressed against and destroyed each other, so that an enormous number of the capillaries which form a network in the walls of the alveoli must have been entirely destroyed, thus limiting the passage for blood through the lungs. Further we notice scattered through the lungs, but especially at the apices, a quantity of black, thready material condensing the lung-substance. On microscopic examination we see that this thready material is thickened connective tissue surrounding the capillaries, the alveoli, and the bronchioles. We are less surprised to find this fibroid degeneration of the lung when we find how commonly emphysema is associated with fibroid disease of other organs. What are the usual coincident changes when a patient has died of vesicular emphysema? We usually find one or more of the following conditions: granular contraction of the kidney (the common association of emphysema with hypertrophy of the left ventricle has long been noticed), the early stage of hepatic cirrhosis, pigmentation, and fibroid thickening of the wall of the stomach, fibroid thickening of the pia mater, fibroid changes in the brain and spinal cord, and atheroma of the arteries (explaining the common association of vesicular emphysema both with cerebral hæmorrhage and with disease of the aortic valves). All these diseased alterations have to be counteracted. To do this, keep the patient as much as possible in the great circulations of light and air, give him sufficient food, and promote excretory action.

Owing to the obstruction to the blood flowing through the lungs, the right side of the heart gets excessively distended, and signs of venous congestion of various organs come on. There is venous congestion of the kidneys. The urine becomes scanty, high-coloured, and loaded with lithates, and there may be uric acid or other gravelly deposits. Venous congestion of the stomach leads to dyspeptic symptoms, venous congestion of the bowel to piles, venous congestion of the brain and cord to disturbance in the cerebro-spinal functions. The patients suffer from mental depression, desire and appetite fail; a peculiar nervousness steals over them, restlessness alternating with drowsiness, and they are commonly subject to attacks of spasmodic asthma, especially in the night. Similar effects occur with severe nervous shock or other great irritation, as, for instance, where there are compound fractures, or severe burns. Again, with much mental anxiety and worry patients commonly say that they are sleepless, and their breathing is affected; we are obliged, therefore, in emphysema, to take into account the vis nervosa. We must endeavour to strengthen the nervous energy of patients, to guide them to be much in the open air. Experience shows that they usually get weaker, with increasing suffering of many kinds, if not enough out of doors.

As the venous congestion increases, there is commonly œdema of the lower extremities, and at length general œdema. The most extreme general œdema I have ever seen was in a case of vesicular emphysema.

I have seen many cases teaching this most important fact, that so long as the right side of the heart does not become excessively distended and dilated, persons may live comfortably with extreme emphysema of the lungs. On the other hand, there are some persons with comparatively little emphysema, but they are fat; they have big bellies, and large accumulation of fat under the skin; they are sluggish in the bowels and the kidneys, and they turn livid; dilatation of the right ventricle comes on early, and in some cases the dilatation comes on the more rapidly because the fat grows in and replaces the muscle of the ventricle. As the circulation in the lungs becomes difficult, the air circulation in the stomach and intestines becomes difficult also, so that they become excessively distended with gas, and such patients often come to the doctor complaining of wind. Such persons suffer from what Dr. Ramskill used to speak of as " the three F's," fat, fæces, and flatus. We must help them to keep their bowels open, for this will relieve the breathlessness from which they suffer so much. The rectum is a breathing apparatus, and we must get the wind down to free the diaphragm and ease the right side of the heart. Here the use of asafœtida comes in ; and you will often here me in the wards order them a Spanish onion.

Let us go on to consider the relation of vesicular emphysema to bronchitis. It used to be taught (and still is) that vesicular emphysema is caused by bronchitis. Why was that taught so dogmatically ? Because emphysema is very often found together with bronchitis, and emphysematous patients very commonly die of bronchitis or broncho-pneumonia. In the course of experience, however, I observed many persons with emphysema and no bronchitis. I asked them, " Have you ever suffered from bronchitis ? " and many said " No." I went further, and found that in some cases of extreme emphysema the patients had suffered little from bronchitis. At last I came to recognise that this degeneration of lung tissue was a part of general tissue degeneration. Vesicular emphysema, or rather the tendency to its early onset, may be an inherited condition ; it is thus met with in certain families, and may attack the young members of those families. This probably accounts, to some extent, for severe forms of emphysema occurring in connection with whooping-cough. If it were due to the whooping-cough alone, it would, we should think, be much more commonly met with in early life.

There is a relation between gout and emphysema, and the children of gouty parents are liable to emphysema, that is, to premature lung degeneration. Emphysema is much more common in the middle or more advanced periods of life, and we may therefore plausibly say that the cause of emphysema usually is the wear and tear of many years. You may be asked, " Don't you think that blowing wind instruments would cause emphysema?" Answer, "Yes, if badly blown." Again, "Don't you think that excessive expiratory and inspiratory actions may cause emphysema?" You may fairly answer, "Such increased air-tension is doubtless the immediate cause of the dilatation of the air-cells, but will only produce emphysema when the tissue of the lung is already in a degenerating condition."

Our ordinary experience of the relation between emphysema and bronchitis is, that as the expiratory sounds in the lungs become more prolonged we find from time to time signs of bronchitis. The bronchial inflammation may be preceded by attacks of catarrh of the windpipe. The earlier attacks of bronchitis are not usually severe, and affect only the larger tubes, but as the degenerative process in the lungs advances it becomes evident that the circulation in the bronchial mucous membrane is becoming weaker and weaker, and that the membrane is becoming less and less able to withstand cold and damp. Damp is more hurtful than cold, and if there is a moist summer the patient may suffer from bronchitis as much as in winter. Each winter the attacks are more severe, as the lung further decays; and the muscular power of the tubes becoming weaker, they are liable to dilate. The power of expelling the muco-purulent secretion fails more and more, there is increasing crepitation throughout the lungs as they become more and more choked, and what is expectorated is less charged with air-bubbles than formerly. The patient may say, "You need not give me anything for my cough, as it is nearly gone," and yet there is more prostration, so that he is able to do less and less. The crepitant râles become smaller and smaller, and the expectoration more like pus than phlegm, and it is almost airless. These signs show that the patient is about to die of capillary bronchitis. But even then, if the right ventricle of the heart can send more blood through the lung, expectoration may increase and the patient rally. Sometimes a few nights' rest will relieve him wonderfully; sometimes the administration of spirits and hot water, and some expectorants, will give him much relief. The most gloomy signs are, loss of cough with diminished expectoration, the pus containing little or no air, and the crepitation less audible until completely lost. I went to a patient with emphysema and put my ear to his chest. I heard no crepitation. In a day or two I was told he was dead. I was puzzled until I saw that there was no crepitation because he was too weak to get air into his lungs.

On making a post-mortem examination in these cases, we find the minute bronchi choked with pus, and we may in some cases further notice that the bronchial tubes are much dilated, especially those near the surface; the dilatations may be lengthwise, reaching to the surface of the lung, or spherical and moniliform. We observe also that the transverse muscular fibres of the dilated bronchial tubes are much thicker in some parts than in others, in which they may be so thin that they are exceedingly difficult to find.

Now let me sum up for you the conditions which in vesicular emphysema hinder the circulation through the lungs.

1. Loss of elasticity in the lung.

2. The lung is unable to expel the air sufficiently, and consequently becomes more and more choked with residual air.

3. The capillaries of the lung are by degrees destroyed by atrophy and fibroid thickening; thus is established a permanent hindrance to the circulation through the lung.

4. As the nutrition of the bronchial mucous membrane fails and bronchitis comes on, the air is more and more shut out from the alveoli by the increasing bronchial exudation.

5. The right ventricle becomes dilated, so that less blood is pumped into the lungs.

6. The movements of the chest are curtailed by the ossification of the rib cartilages and of the intervertebral substances.

7. The muscular and nervous powers of the body are more and more weakened as oxidation fails.

8. As these changes slowly creep on, the tissue of other organs undergo fibroid degeneration—the kidneys, the liver, the stomach, the brain, &c.

Thus we learn that in studying emphysema we must recognise other conditions than the statical condition of the lung. We do not breathe by lung-substance, for this is merely the framework; we breathe by blood, so that the condition of the blood in emphysema is a most important matter. If the blood becomes anæmic, then the lung become much atrophied. Always, when thinking of blood, go right down and think of water, which is the great basis of blood. If you watch cases of cholera you will notice this—that as the blood gets thicker and loses its water, the breathing fails more and more. The blood is thick and tarry, and the patients are craving for water.

LECTURE XXVI.

BRONCHITIS.

It is usual to speak of three varieties of bronchitis, according to the seat of the inflammation—bronchitis of the larger tubes, bronchitis of the medium-sized tubes, and bronchitis of the tubes which are so small as no longer to have plates of cartilage in their walls, or capillary bronchitis. We also speak of acute and of chronic bronchitis.

It is useful to know that inflammation of the larger bronchial tubes is attended with little or no danger, provided the general circulation be not failing. Inflammation of the larger bronchial tubes occurring with typhoid fever, and in other asthenic states of the system, may be a source of great danger. The accumulation in the bronchi may be enough to prevent a weak patient from getting air into the lung.

Bronchitis becomes dangerous in proportion as the smaller tubes are affected. With bronchitis attended by much harassing cough and expectoration there is usually no danger, and the same may be said when the expectoration has a large quantity of air in it.

If asked, " What form of bronchitis kills ? " say " Capillary bronchitis." " How do you know that ? Would you find it out on post-mortem examination by opening the larynx, trachea, and larger tubes ? " "No ! I should cut across a piece of lung —at the base by preference—and squeeze it and see the pus oozing from the smaller tubes." " How would you demonstrate that this had killed the patient ?" " By finding that as the air was shut out from the tubes by pus (the secretion is always purulent in capillary bronchitis) the blood was cut off from the left side of the heart. I should find the left ventricle empty and firmly contracted, the right side of the heart and the veins distended."

If you are asked, "What do you mean by bronchitis?" say, "There is hyperæmia of, and exudation from, the capillaries of the bronchial mucous membrane; an exudation of an albuminous mucous substance swelling up the epithelium, and thus destroying it more or less; the nuclei of the epithelium divide and become mixed with leucocytes and form pus; the muco-purulent matter is expectorated."

What are the appearances of the bronchial mucous membrane in cases of bronchitis? It may be, and commonly is, exceedingly congested with hæmorrhages—these are the usual appearances of severe catarrhal inflammation. The epithelium is detached and the membrane granular, having much exudation in its substance, which gives it a swelled and dull appearance. Is this morbid change a recoverable condition? Certainly. Does it tend to ulceration? No, except where the tubes are dilated, or where there is phthisis, cancer, or some other grave condition. Where the tubes are dilated, or where there is extensive suppurative bronchitis, the bronchial mucous membrane may be destroyed by sloughing. This may also occur in syphilis (rarely), and in pyæmia. Should we be surprised to find sloughing where the patient was suffering from nervous failure? No. I have found gangrene of the lung in connection with epilepsy, but what I am especially referring to here is diabetes. In this disease, instead of carbuncle or of sloughing of the toes, the gangrenous changes which so often come on may attack the lung.

What are the antecedent conditions of bronchitis? Do not let us use the word "cause," for it produces fogginess. What we want to know is what went before and determined the bronchitis.

1. Degeneration of the lung-substance is commonly attended by bronchitis; hence bronchitis is usually present in vesicular emphysema, and in the various forms of fibroid degeneration of the lung.

2. Morbid growths in the lung substance, notably tubercle (acute tuberculosis usually kills by capillary bronchitis), cancerous growths, hydatid disease, syphilitic changes. In

cases where there is bronchitis and no obvious cause for it can be found, suspect old damage of the lung.

3. Another antecedent condition is dilatation of the bronchial tubes.

4. Another cause is congestion of the lungs in heart-failure, and this is more liable to lead to bronchitis if there is any slow process of degeneration going on in the lung itself; thus bronchitis is common in cases of dilatation of the left ventricle in which there is more or less fibroid degeneration of the lung.

5. Congestion and œdema of the lungs may be due to failure of nerve energy, so that bronchitis is liable to come on in all kinds of brain failure. One of the great risks in hæmorrhage and other cerebral disease, and in injuries to the head and spine, and in disease of the spinal cord, is from bronchitis passing into pneumonia. Similarly, in mental diseases there is danger of bronchial catarrh passing on into what is called phthisis.

6. Excessive accumulation of serum in the lungs in Bright's disease may give rise to rapidly fatal bronchitis. You may see a patient sitting up in bed struggling for breath, and on listening to the chest you hear mucous râles everywhere. You look at once to see what he is expectorating, and you find it is froth. On examining the urine you find albumen; and there are other signs of Bright's disease. In a case like this it is of no use to waste time in giving squills. One evening I gave a man dying of œdema of the lung a drop of croton oil. He was severely purged and was comparatively well the next day.

In connection with this do not overlook the fact that there may be extreme struggling for breath, and no signs of bronchitis to be heard for some hours, whilst œdema of the lung is rapidly coming on. The breathing is extremely laboured, but when we listen to the chest we hear no abnormal breath-sounds, we only notice that the sounds are rather faint, but the air is certainly going into and out of the lung. In this stage there is congestion and œdema of the lung-tissue, but as yet no exudation into the tubes.

7. Foreign bodies getting into the bronchial tubes, as

food in cases of œsophageo-tracheal fistula, may set up bronchitis. Scalding, by inhalation of boiling steam is another cause.

8. Poisoned blood brings about bronchitis, so that it is common in measles, typhoid, and various other fevers, but it is rare in scarlet fever. A patient came to the out-patient department for cough. There was bronchial catarrh, but no heart disease or other obvious cause for it. The temperature was a little raised, and I admitted him, suspecting typhoid fever, and this it proved to be.

9. Bronchitis may arise by extension of inflammation from the larynx or trachea. This often occurs in croup, in diphtheria, and in syphilitic disease of the larynx and trachea.

10. Inflammation may extend from the pleura or parenchyma of the lung to the bronchial tubes. Is cough a symptom of pleurisy? No. Then when cough accompanies pleurisy, what do we infer? That the inflammation has extended to the bronchial mucous membrane. We see a case in which there is great effusion into the pleura; then a severe cough comes on, and there is expectoration of pus, and we think, perhaps, that the effusion in the pleura has burst into a bronchial tube. Nothing of the kind has occurred, but a severe bronchitis has been set up by extension. If an empyema had penetrated the lung and opened a bronchial tube, there would of course be pneumothorax.

11. I know nothing about idiopathic bronchitis. I know nothing about bronchitis produced by cold in healthy people. Some would ask, "Do you not find primary bronchitis in children?" Where I have found bronchitis in children I have usually been able to trace rickets, struma, or tubercle. Further, you might be asked, "Does not bronchitis usually prevail in the wet and cold weather of spring and autumn, and is it not produced by these conditions of the weather?" Yes; no doubt that is so. But it is in persons who have clear evidence of other disease.

What do we understand by chronic bronchitis? We understand a bronchitis which recurs again and again, usually every spring and winter, for years. Let us consider the conditions which give rise to this recurrence. When after death we examine such bronchial tubes, we usually find them much thickened; their mucous membrane has lost its transparency, and is more opaque and granular; its epithelium is much destroyed. On microscopic examination, instead of seeing columnar epithelium, we see a number of imperfect epithelial cells, with pus cells and much granular matter. We find that the thickening extends into the sub-mucous tissue; in other words, that the capillaries of the mucous membrane are thickened, and doubtless the serous passages blocked to a great extent. It is almost inconceivable that the capillaries can be thickened and yet the areolar spaces around them not be blocked. For this reason the serous circulation must be weak, and liable from time to time to be much impeded, and it may be completely arrested. This being a weak spot in the body, if the general serous circulation becomes embarrassed and disordered, then the weak bronchial circulation will be the first to fail. Thus we can understand the influence of cold and wet; for the serous circulation is very liable, when weak, to fail in cold weather. Further, think of the serum. It is mostly water, and the water in the serum and the tissues may be regarded as one continuous whole. In evidence of that, let me remind you that when a man is attacked with acute diarrhœa he shrinks from head to foot, or with profuse sweating he shrivels. We can understand that when cold weather and east wind lessen the function of the skin and the escape of water from the lungs, there arises difficulty in the serous circulation.

If these things are considered, it becomes obvious that we may fail to protect persons from the effects of east winds even by keeping them in bed; and, in fact, experience has shown that bedridden persons are susceptible to the influence of east wind.

All this becomes of immense importance when we have to treat these cases. We have to learn that the more we keep them indoors so much the more sensitive they become to changes of weather. To be much in the open air will strengthen the nervous energy of the patient; and a healthy vis nervosa and a healthy working of the serous circulation go together.

LECTURE XXVII.

DILATATION OF THE BRONCHIAL TUBES

The part of the tube always affected by dilatation is where it ceases to have its wall strengthened by cartilaginous plates, and becomes entirely membranous. We determine whether the tubes are dilated by cutting them open. If they are dilated, the further we pass the more readily the point of the scissors slips along, the tube being wider near the pleura than an inch or so higher up. Sometimes we come across a little sac. In the lesser degrees of dilatation this is not the case, but still we are able to open the minute tubes very readily; we can slit them open right up to the end.

There are then two forms of dilatation, uniform and saccular. In the first there is a uniform swelling from the point where the cartilage ceases down to the end of the tube. If the wall of a tube bulges here and there to form a little cavity, we have saccular dilatation. The two forms commonly coexist. Sometimes, in addition to this minor degree of dilatation, we find one or more large cavities, the size of a walnut or even of a small orange. Such a cavity has a distinct boundary of a tough kind, and we notice in the wall transverse fibres which look like the unstriated muscular fibres of the bronchial wall; then we are sure it is a dilated bronchus. Such cavities occurring in the upper part of the lung have been diagnosed as phthisical, but the disease commonly extends over many years and has other peculiar characters, enabling us to distinguish it from phthisis.

On looking at the bronchial mucous membrane in these cases what do we find? Thickened mucous membrane, thickened submucous tissue, with fibroid deposits. The membrane is granular and pigmented, owing to chronic congestion; it is commonly covered with pus. Owing to the thickening, owing to the repeated congestion, the nutrition of the mucous membrane becomes weaker and weaker, so that it is liable to slough. In some parts the transverse muscular bands stand out very distinctly, but in others they are lost. Here, as elsewhere in the system, we commonly see hypertrophy and atrophy side by side.

Owing to the atrophy of the muscle and to the loss of elasticity in other ways, the bronchial secretion tends to accumulate more and more. It becomes more and more purulent and it decomposes. Thus in this disease our attention is first riveted by the extremely offensive smell of the sputum. As the sputum decomposes it loses the yellow aspect of pus and becomes sea-green; if still more decomposed, a grey, watery substance is expectorated. The exudation of pus is usually large, causing much expectoration day by day. The accumulation of decomposing pus is liable to set up suppurative pneumonia, and the inflammation may go on to sloughing, so that sloughing cavities are formed beside the dilated bronchi. Owing to the prolonged congestion, thrombosis is apt to occur, and then at the post-mortem examination we find appearances like those of pulmonary apoplexy in the neighbourhood of the dilated bronchus. One of the symptoms of thrombosis in the vessels of the lung is hæmoptysis, and I have known patients with dilated tubes die of such hæmoptysis.

The outer coat of the bronchial tube is much thickened by fibroid tissue, and this thickening spreads and fills the air-cells, so that we get fibroid consolidation of the lung in connection with dilated bronchi. Now which was the prior change? Did the dilatation of the bronchi lead to chronic inflammation, and this to the fibroid thickening, or was the fibroid consolidation the first stage in the process, the subsequent contraction of the fibroid tissue having drawn open the bronchi? In our decision we should be guided by the following considerations. In cases in which there is a very large amount of fibroid tissue, as in the so-called cirrhosis of the lung, in which disease we usually find some amount of dilatation of the tubes, we have no doubt that the dilatation of the tubes is secondary to the fibroid change. In other cases, in which the fibroid thickening is limited to the dilated bronchi and a few adjoining air-cells, we have no hesitation in saying that the primary change is the bronchial dilatation.

Under what circumstances does dilatation of the bronchi occur?

1. In connection with cirrhosis of the lung.

2. When the air-cells are filled up by morbid deposit so that the elasticity of the lung is lost. Thus some degree of bronchial dilatation is common in phthisis, between the consolidated areas, but the dilatation is not great.

3. Since loss of elasticity in vesicular emphysema is very marked, bronchial dilatation is often found in association with this disease.

LECTURE XXVIII.

PLEURISY.

I WILL begin this subject by describing the morbid anatomy of pleurisy. The first thing we notice on examining the pleura is the loss of brightness. The membrane has lost its bright translucent appearance and is dull and opaque; it is no longer smooth, but is covered with a fine granular matter. If the patient has died in the acute stage we find the pleura extremely congested. In many cases, however, the acute stage is over, so that the congestion has passed away, or is concealed by the lymph which has been poured out. The pleura is much swelled, and on microscopical examination we see that the fibres of the membrane are swelled up and separated, a large quantity of leucocytes having been extruded between them. If the inflammation has been very acute we find red corpuscles as well as white. We can estimate the severity of a pleurisy, as of other inflammations, by the number of red corpuscles we find lying in the inflamed tissue. Therefore the most severe and most rapidly fatal form of pleurisy is the so-called hæmorrhagic pleurisy, in which the inflamed membrane looks like a tough blood-clot, and the serum in the pleural cavity is stained with blood.

What are the common terminations of pleurisy?

1. Simple pleurisy does not kill the patient. I never made a post-mortem examination on a patient who died from simple pleurisy; therefore if there is pleurisy without any antecedent severe disease the patient recovers. One termination then is entire recovery. I cannot tell you how recovery occurs, for as you see I have had no means of finding it out.

2. The natural tendency of a pleurisy is for the lymph to

become organised. Cellular tissue is formed and the two sides of the pleura become united. This is termination by adhesion, and is the commonest ending of pleurisy. The adhesions may evidently lie harmless for years and years. How is it that they are harmless? Because they become vascular, and are able to work easy with the general circulation. If they were not vascular they would be a dead hindrance. But it is notorious how little disturbance is caused by adhesion of the pleura.

3. Not only may there be adhesion but the pleura is liable to become much thickened. This is termination by fibroid thickening. But even where the pleura is very much thickened by fibroid tissue it becomes more or less vascular, and may be harmless for many years. But inasmuch as these vessels that are formed to carry on the circulation in the new tissue are buried in fibroid thickening, they are evidently weak vessels, and the circulation through them is bad, so that if the general circulation is weakened from any cause these vessels are liable to fail early. Hence these old fibroid thickenings are very liable to recurrence of inflammation, and at the post-mortem examination we often find recent acute pleurisy around old fibroid thickenings. Are we to apprehend any other ill effects in fibroid thickening of the pleura? Yes, it may extend more or less into the lung, producing fibroid consolidation. Further, the thick fibroid tissue may contract and compress one or more lobes of the lung; I have seen it cause almost complete collapse of a lung.

4. Serous effusion into the pleural cavity may occur. We find in the pleural cavity a clear yellow albuminous fluid, containing flakes of lymph. What are the effects of such effusion? Need we apprehend much evil from it? No; certainly not for a time. But suppose that a large effusion greatly compresses the lung, is there any cause for alarm? No; as long as there are no symptoms of serious respiratory difficulty we need not be alarmed for two or three weeks. What are the actual results of the effusion? The lung is more and more pressed towards the vertebræ, the air is squeezed out, so that the lung becomes compressed and sinks in water. It is pressed away from the front of the chest and from the axilla,

inwards and backwards towards the vertebræ. It is important to remember this fact in clinical examination. We may still hear the air entering the back part of the lung—the sound is extremely tubular—and there is increased voice and increased tactile vocal fremitus, and we may mistake the signs for those of pneumonic consolidation; this mistake is easily avoided by noticing the complete absence of breath-sound in front and at the side of the chest.

We next have to ask how long the effusion may remain without causing permanent damage to the lung. Clinical experience shows us that it commonly disappears in two, three, or four weeks, leaving no appreciable damage. For a few weeks, then, the presence of a large quantity of fluid in the pleural cavity may be tolerated with impunity; but, after a few weeks, if the fluid is not removed, the pleura is liable to become thickened, and to contract, and so permanently to compress the lung. You will see the important bearing of this experience when I come to discuss the treatment of these conditions.

5. Pleurisy may end in suppuration; but this is a form of pleurisy of a special kind. By this I mean that the termination of a pleurisy in suppuration is due to the existence of a special cause of suppuration, and therefore we may say that suppuration is not the natural end of pleurisy. What these special causes of suppuration are, I will discuss later.

What are the causes of pleurisy? If I ask a student this question he always begins with "Cold." I say to him, "How do you know that exposure to cold causes pleurisy? We know that pleurisy occurs in connection with Bright's disease, we see it in the wards and on the post-mortem table. But what do we *know* about the relation of cold to pleurisy?"

I consider it very doubtful if cold can excite pleurisy in a moderately healthy subject. When we are told that pleurisy has followed taking cold, or exposure to cold, we usually find that there is some other morbid condition associated with the pleurisy. If we think too much about cold, we may overlook rheumatic fever coming on, or pneumonia, or phthisis, or one of the many other morbid conditions with which pleurisy is associated. When I see a patient with pleurisy I never make up my mind that the cause was cold, but I leave it an open question as to what may be coming on. Why do I do this? I have had so many warnings. I have known many patients with pleurisy, and apparently nothing else—and in one case the patient died of phthisis shortly afterwards, in another a day or two later we could hear a loud mitral murmur, and in another it was not long before we had evidence of cancer growth in the chest.

You will be asked, "Is there such a thing as idiopathic pleurisy?" The answer is simple: "Idiopathic pleurisy is pleurisy of which we do not know the cause." To one another, and even to the public in these days, we can sometimes admit that we are ignorant; but "idiopathic" was a word much clung to.

If you are pressed, you will do well to admit that pleurisy *may* be due to exposure to cold, but you should add that we know nothing about it, and that in such cases as appear to have arisen in this way it is probable that extreme nervous or physical fatigue had an important share in the causation.

What are the causes of pleurisy? You know that though I put the question in this common way, what I really mean is, "With what morbid conditions do we find pleurisy associated?" These morbid conditions are very numerous.

I. Simple pleurisy is very commonly connected with a tendency to rheumatic fever. I am not speaking now of the cases of pleurisy which occur during a rheumatic outburst, but cases in which the subsequent history, or the previous history, or the history of other members of the same family, proves the existence of a tendency to rheumatic fever.

II. One of the commonest associations of simple pleurisy is with the tendency to phthisis. A patient has an attack of pleurisy, there are no signs of phthisis, but from the conformation of his chest, or from his family history, we are led to think that he has a tendency to phthisis; very often in such cases the patient has well marked phthisis in a year or two.

III. A mild form of pleurisy sometimes comes on in the course of secondary syphilis. This brings me to speak of other kinds of blood-poisoning in which pleurisy is likely to come on.

The conditions of infection of the blood and serous system in which pleurisy may occur are:

1. Various fevers, notably secondary syphilis, scarlatina, and measles. In typhoid fever it occurs, but rarely. The pleurisy which occurs in connection with ordinary lobar pneumonia should probably come under this head, but it might be classed as arising from extension of inflammation.

2. I told you that pleurisy is often a sign of the rheumatic tendency, but further it is exceedingly likely to arise in the morbid state of the blood present during a rheumatic outburst, in this respect the pleura resembling the pericardium. Rheumatic fever may be ushered in by pleurisy or pleuro-pneumonia before the pericardium or the joints are affected. If a young subject has pleurisy and there is no sign of phthisis, always think that perhaps rheumatic fever is coming on. Often in rheumatic fever the inflammation begins in the pericardium and extends thence to the pleura.

What part of the pleura is commonly affected in rheumatic people? The portion of the left pleura most in contact with the pericardium, over the lower and inner part of the left lung. When this piece of pleura, which is in contact with the apex of the heart, is affected, the signs are rather puzzling. We hear what appears to be a pericardial rub at the apex of the heart, but we notice that the sound is regularly intensified with each inspiration, and this tells us that its origin is without the pericardium. As the lung is inflated the two roughened pleural surfaces are rubbed together more forcibly by the movements of the apex of the heart than when the lung is collapsed in expiration, hence the inspiratory augmentation of the sound.

3. One of the most common pathological antecedents of pleurisy is Bright's disease. Pleurisy is common in cases both of granular kidney and of large white kidney, and also in acute nephritis, where there is much general œdema and œdema of the pleura. Pleurisy is especially common in cases

in which there are uræmic symptoms, cases in which the blood is loaded with excrementitious products.

4. In thinking of poisoned blood we must not forget the presence of bile in the blood, and we are never surprised if pleurisy comes on in the course of jaundice.

5. The most serious of all the morbid states of the blood with which pleurisy is associated is pyæmia. In such cases the pleurisy is usually purulent, and I shall return to consider them later.

IV. Pleurisy may arise from injury. Pleurisy, and very severe pleurisy, may be produced by a blow on the chest without any fracture of the rib, and without any external wound. I once made a post-mortem examination on the body of a young muscular man who died rapidly from suppuration of the pleura, after a blow on the side. There was no other lesion. This will teach you, in the puzzling cases of empyema you will sometimes meet with on the post-mortem table, in which all the other organs are healthy, to take into consideration the possibility of trauma. In this connection I may remind you that we may have severe and fatal contusion of internal organs without any external sign of injury, and without any fracture of bone.

V. Morbid growths in the lungs commonly lead to pleurisy.

1. The most common is tubercle. If asked for the common causes of pleurisy, begin with tubercular deposit in the lung. In many cases the tubercular deposit is too small to be revealed by objective signs, but pleurisy is set up, and in a few weeks the signs of phthisis may be unequivocal.

2. Cancer of the lung, like tubercle, often first attracts our attention by setting up pleurisy.

3. Tertiary syphilitic deposit in the lung may excite pleurisy. More common than gummata, is scattered syphilitic thickening of the lung and pleura, and in this thickened tissue acute pleurisy is liable to arise.

4. In emphysematous lungs, the pleura is more or less thickened, and the vessels are weak, and attacks of pleurisy are not uncommon; the same may be said of heart disease, and of other conditions in which there is chronic venous congestion.

VI. Pleurisy may arise by extension of inflammation from other organs.

1. The most typical example of this might be thought to be the pleurisy in cases of pleuro-pneumonia, but this is rather an associated than an extending inflammation.

2. Rheumatic pericarditis, or pericarditis from other causes, frequently sets up pleurisy.

3. Inflammation may extend from the liver, but this is a more common cause of empyema than of simple pleurisy; a suppurating hydatid bursts into the pleural cavity.

4. Caries of the vertebræ may excite pleurisy. This may be in a case in which there is no gross change in the bones to produce deformity. Therefore, whenever there is protracted fixed pain in the back, followed by what appears to be simple pleurisy, think of the possibility of the cause being a localised caries of the front of one of the vertebral bodies. Similarly caries of a rib, or of the clavicle or sternum, may set up pleurisy by extension.

5. Pleurisy sometimes comes on in cases of aneurysm of the aorta.

6. Strumous mediastinal glands.

7. Lymphadenomatous glands.

8. Any form of broncho-pneumonia may determine pleurisy, and we must recall some of the common causes of broncho-pneumonia. Therefore in syphilitic laryngitis, in cut-throat, in laryngeal diphtheria, in scalds of the glottis, &c., we are never surprised by the onset of pleurisy.

9. Inflammation may spread through the cellular tissue to the pleura. It may thus arise after tracheotomy, or after operations at the root of the neck, such as ligature of the common carotid. There is one variety of which you must be well aware. In some cases of suppurative disease of the middle ear, the inflammation may spread down along the sheath of the internal jugular vein, and through the cellular tissue to the pleura. You would suspect that this extension was taking place, if in a patient with middle ear disease, there were pain and stiffness in the neck, with deep induration over the course of the vessels.

I pass on to consider the causes which are especially liable to produce suppurative pleurisy. The cause is very commonly suppuration in the lung itself, and the conditions in which the latter is most commonly met with are phthisis and pyæmia.

1. Pyæmia leads to a very fatal form of pleurisy; a small abscess forms in the lung just under the pleura, and tends to burst into the pleural cavity; over this area a quantity of lymph is formed—an endeavour of nature to prevent the perforation—and we find on post-mortem examination that this lymph is yellow, and charged with pus, or we may find a large quantity of pus in the pleural cavity. Some cases of pyæmia are very obscure, and the only objective signs may be those of pleurisy. If in a patient who has been exposed to one of the causes of pyæmia we find that a pleurisy develops, we fear that the case may turn out to be one of pyæmia.

2. Incipient or threatening phthisis causes simple pleurisy; it is in cases of confirmed phthisis with disorganisation of the lung that we meet with empyema. There is a small vomica situated, like the pyæmic abscess I have just described, immediately under the pleura, and this may burst into the pleural cavity, leading to suppurative pleurisy with pneumothorax, but these cases are rare, and more commonly we find no air, but pus, mixed however with adhesive lymph, indicating some attempt at repair. Phthisis is by far the commonest cause of empyema, and therefore in every case of empyema you should examine carefully for phthisis.

3. We sometimes meet with empyema in cases of cancer of the lung.

4. Caries of the vertebræ, or of the chest-wall.

5. Injury, such as fractured rib, but especially penetrating wounds.

6. A suppurating hydatid may burst into the pleural cavity.

7. A liver abscess may take the same course.

8. Empyema is very common in Bright's disease. It may arise spontaneously, but often follows the operation of paracentesis. In Bright's disease there is a special liability to suppurative inflammations after slight injuries.

9. In some cases of empyema no traceable antecedent can be found. These cases are exceedingly rare, and experience

has led me to think that there has been in operation some cause of profound exhaustion, which has materially impaired the nutrition of the tissues.

We will now consider the suffering and signs of pleurisy. In rare cases the onset is insidious, and there is little or no appreciable suffering until the breathing becomes seriously embarrassed by the bulk of the effusion. This occurs only in cases in which the effusion is poured out very slowly and in which the inflammation of the pleura is never very acute. We meet with such conditions in Bright's disease and in phthisis. You will not then be surprised if a patient comes to you complaining only of shortness of breath, and on examination you find that one side of his chest is full of fluid. Such cases are by no means rare. A man may walk about with over a hundred ounces of fluid in his pleural cavity, and keep at his work for weeks. The disturbance of the general circulation in pleural effusion is in proportion to the rapidity with which the effusion is poured out, and in these cases it is formed so slowly that the heart has time to get accustomed to the displacement. A very important question in cases of pleural effusion is, when are we to interfere? We are not to interfere unless urgent dyspnœa calls for it. I have known a patient tapped during the earlier inflammatory stage when there was only a few ounces of fluid to remove, but that was because his breathing was much embarrassed, and we hoped to relieve it. The only other condition in which it is necessary to interfere is when it becomes evident that there is no tendency to absorption, but that the fluid is likely to remain and to cause permanent damage. Remember that thickened pleura may give signs very similar to, and sometimes even indistinguishable from, those of pleural effusion, and the same may be said of certain cases of malignant disease of the lung.

I meant, however, to speak of the suffering and signs in ordinary acute pleurisy. Pain is commonly the first indication—a severe stabbing pain which makes the patient dread to breathe. We estimate the severity of an attack of pleurisy by the severity of the pain, for if a patient dies in the early stage of a pleurisy it is the pain that kills him. With the pain there is commonly some degree of fever, and this fever may be a guide if you are in doubt whether the pain is due to pleurisy or to nerve-irritation, for as yet there is no other sign of pleurisy than pain. Remember this, that the pain is most severe before we can hear any rub, and that as the rub becomes audible the pain passes away. We believe that the pain is due to the excessive stretching of the tissue, due to the vascular engorgement which is the first stage of inflammation, and that the relief of the pain is due to the relief of the tension which ensues on the pouring out of the exudation. Now the rub is due to the exudation, and thus we can easily understand the course of the symptoms.

As the pain increases the pulse becomes small and hard, and we measure the danger by the condition of the pulse. The condition of the pulse is due to the pain and irritation, which act in two ways: first, the action of the heart is depressed, so that the blood accumulates in the right side, and only a very small quantity of blood is thrown into the arteries at each systole, hence the smallness of the pulse; second, there is a contraction of the peripheral arteries, raising blood pressure and making the pulse hard. The face becomes strained and set, and the breathing becomes difficult. Pain kills by depressing the circulation and the breathing. The breathing becomes laboured, the blood cannot get through the lungs, and lividity comes on. As the breathing fails more and more you will recognise it by finding that the stomach feels hard and tight when you pass your hand over it. When the circulatory failure is extreme there may be vomiting from congestion of the stomach, and hiccough is not uncommon. Hiccough, when persistent and severe, is a symptom of grave depression.

Now, can the patient recover from such a desperate condition as this? Yes. What are the indications for treatment? Relieve the pain when severe at any cost. You will be guided as to whether the patient is improving by the pulse becoming freer, the countenance less blue, and less set. Now the pain is relieved when the tension is relieved, and how are we to relieve the tension? Opium relieves the consciousness, but leaves the tension unaffected; to relieve this you must have recourse to local measures, such as leeches, dry cupping, or a poultice. Do not be content with merely giving opium until the pain is no longer felt—you may stupefy the patient with opium, and the breathing may rapidly fail.

How are we to tell when there is pus in the pleural cavity? We can only be certain if we have seen the pus, by drawing off some of it with an aspirating syringe. We may suspect it where the temperature is very irregular, or of the hectic type, and in all cases of doubt a syringe should be used to clear up the question. A consideration of the cause of the pleurisy is important; for instance, rheumatic pleurisy is never purulent, and the pleurisy of Bright's disease is rarely so, unless tapping has been performed, whilst empyema is common in phthisical patients, after injury, and in cases of pyæmia. Further we should remember that empyema is more common in children than in adults.

What have we to apprehend in empyema? It may, and not rarely does, kill the patient at an early stage. But more commonly life is continued until there is a large accumulation of pus in the pleural cavity. Sloughing of the pleura is very rare except in conection with pyæmia. Where empyema persists for a long time it commonly leads to much thickening of the pleura, and the thickening of the pleura is followed by contraction, leading to much deformity of the thorax. Here is one reason for letting out the pus as soon as possible.

Pus in the pleural cavity tends to stop the circulation; it either does so by leading to rapid collapse, or by increasing asthenia, and for this reason the pus must be removed as soon as possible. Further, pus in the pleural cavity tends to destroy the surrounding structures. It may burst into the lung and much of it be coughed up, while air is let into the pleura, so that we have a pyo-pneumothorax. It may cause fatal pericarditis or peritonitis by extension. It very commonly extends through the intercostal muscles, and may strip the periosteum from one or several ribs, and lead to necrosis. You see how urgent is a speedy evacuation of the pus.

Even a small quantity must be removed without delay, for pus is a terrible irritant, and tends to cause death by collapse. We very seldom are called upon to remove a small serous effusion, but quite a moderate amount of pus in the pleural cavity may kill the patient rapidly. In Charlotte Ward one evening I saw a woman who did not seem to be suffering much. There was a pleural effusion, and the pleura seemed to be about a quarter or a third full. She could sit up in bed, and was not greatly distressed. Shortly after I saw her she began to get worse and was dead in a few hours. At the post-mortem examination I found about a pint of pus in the pleural cavity. There was no other morbid change.

LECTURE XXIX.

PNEUMONIA.

How shall we group cases of pneumonia? This may be done in several ways. We may consider the part of the lung affected, and so get an anatomical grouping. We may divide the cases according to the antecedent or cause. We may group the cases according to the direction from which the inflammation comes.

Anatomically, pneumonia is grouped into—(1) Lobar pneumonia, where a large area of lung is inflamed. (2) Lobular pneumonia, where the lung is affected in much smaller areas, the appearance it presents being due to the form of the lobules. (3) Vesicular pneumonia, in which still smaller areas are affected.

Lobar pneumonia very commonly comes on sthenically—that is to say, strongly—and many patients recover quickly. Lobular pneumonia comes on asthenically, and more commonly ends fatally. This is the typical form of pneumonia that occurs in pyæmia and phthisis. Vesicular pneumonia occurs in weakly subjects; it is the pneumonia of children and old people. It occurs also in much damaged lungs, in which there has been old fibroid or phthisical change. Scattered vesicular pneumonia is easily mistaken for acute tuberculosis.

Let us next consider the antecedent conditions of pneumonia. The chief are—(1) Rheumatic fever. Pneumonia is common in rheumatic fever, especially when the heart is damaged. We also frequently see pneumonia in connection with rheumatoid arthritis and gout. (2) Blood-poisoning. We meet with pneumonia in measles, in syphilis, in malaria, and all poisoned states of the system. Whenever a patient is suffering from such poisoning, look out for pneumonia. On the other hand, remember that a poison in the blood may have given rise to an apparently simple pneumonia. I have seen a patient treated for acute pneumonia, and typhoid fever never suspected till the post-mortem examination was made. (3) Morbid growths in the lung. Think especially of tubercle, but also of cancer. (4) Rickets is the chief cause of the vesicular pneumonia of children. (4) Tissue degeneration of the lung is a common antecedent of pneumonia. Pneumonia may come on in cases of vesicular emphysema, or where there is fibroid degeneration of the lung, or where there are remains of old phthisis. Prolonged venous congestion of the lung is a frequent antecedent of pneumonia, and therefore pneumonia and heart-disease are often associated. (6) Neuro-vascular pneumonia. What do we understand by this term? Clinical experience puts it beyond question that extremely sensitive, nervous, and unduly active persons are especially liable to pneumonia. A patient may die of acute pneumonia, and on inquiry we learn that he has been liable to excessive loss of blood from trifling injuries, or that he has been liable to other neuro-vascular disturbances, such as herpes. The history of bleeders, and of persons of a sensitive, quick nature, shows an association with pneumonia. In other cases we may see pneumonia when there is unmistakable evidence of previous excessive expenditure of nervous energy. Thus, an overworked man gets a shock, and acute pneumonia comes on. (7) Gross degeneration of the vessels, such as occurs in Bright's disease, or arterio-capillary fibrosis, is a common antecedent of pneumonia. (8) Another form of pneumonia is apparently connected with death in the blood, such as anæmia. As the red blood-discs become fewer and fewer, there is more and more tendency to pneumonia.

We now pass to consider the third method of grouping the cases, according to the direction from which the inflammation comes. We find on post-mortem examination, in some cases, isolated patches of lung consolidated by pneumonia, and on looking at these solid parts we see in their centres bronchial tubes filled with pus. The clinical history reveals that the case began like bronchitis, as evidenced by the muco-purulent expectoration and by other signs, We therefore consider that the inflammation began in the bronchial tubes, and extended into the air-cells, so becoming broncho-pneumonia. Such pneumonia may occur in patches of various sizes ; and when the patches are very small, limited apparently to a few air-sacs, we may term it vesicular pneumonia, whereas when the patches are larger, but still retain a circumscribed form corresponding to the lobules of the lung, it is what we speak of as lobular pneumonia. These are, then, varieties of broncho-pneumonia. We may find both conditions in different parts of the same lung.

If, on the other hand, the inflammation has spread from the pleura to the substance of the lung, we speak of the condition as one of pleuro-pneumonia. Whilst thus classifying for convenience, let us not for a moment overlook that in some cases inflammation may affect at once pleura, bronchi, and lung-substance, this being a broncho-pleuro-pneumonia. Before describing pleuro-pneumonia it will be well to finish what I have to say of broncho-pneumonia.

Broncho-pneumonia may extend from lobule to lobule, so that a large portion of the lung is consolidated, it may be one or more entire lobes, but remember that this is not what we usually speak of as lobar pneumonia. If we examine the inflammatory deposit of broncho-pneumonia with the aid of the microscope, what do we find ? The air-cells are choked up by inflammatory exudation, consisting of leucocytes, red corpuscles, and granular matter, but, and this offers a marked contrast with the microscopical appearances of pleuro-pneumonia, the interstitial tissue of the lung is comparatively little affected. Now let us leave these anatomical morbid effects, and learn more of the clinical pathological history.

Vesicular pneumonia of children is commonly found in association with rickets. We find it in precocious children, with a tubercular tendency, beautiful creatures that have been made restless and unduly sensitive, until at last they are sacrificed on the altar of wretched conceits. Dickens has depicted such a nature in little Dombey. A precocious child, over-sensitive, over-observant, restless, made exceedingly imaginative, until dreams "murder sweet sleep." These children are restless, their flesh wastes, and their respiration is hurried and abnormal. In watching them from month to month you will soon be impressed that excitement and fatigue are death's agencies.

Such children are prone to catch cold, liable to feverish outbursts, and commonly become the victims of pneumonia. In some families so-called healthy children are disposed to plastic inflammation, such as croup or pneumonia; you may also meet with rheumatic pneumonia, and do not forget that pneumonia in children may be due to measles or whooping-cough. Whenever we attend a child with bronchitis, we have to keep our minds open, and to remember that there is a great tendency for the bronchitis to pass into broncho-pneumonia, and we generally know that broncho-pneumonia has supervened by the much-increased temperature, and by the crepitation having become much finer—more like what is heard with pneumonia in general.

Lobular pneumonia may supervene because a vessel of the lung has been plugged, as in anæmia. It may be due to embolic pyæmia. Some medical men have taught that this pneumonia may be produced by emboli detached from a clot in the right ventricle of the heart. Lobular pneumonia is often due to failure of vis nervosa, therefore we commonly meet with it in brain disease of all kinds.

Lobular pneumonia and asthenic eonditions co-exist. This form of pneumonia occurs in typhoid fever, and in syphilis. It is the pneumonia of old people, of those in fact who are much exhausted. Another form of lobular pneumonia is that which comes on in connection with irritated and inflamed states of the air-passages, such as croup, cut-throat, diphtheria, scald of the glottis, aneurysm pressing upon the air-passage, or cancer in a similar situation.

I have mentioned to you, and often shown to you on the post-mortem table, lobular pneumonia secondary to bronchitis and emphysema, the pneumonia occurring in small patches scattered through the lungs. I may delay you a few minutes by describing the clinical features of such cases. When a patient is suffering from emphysema and bronchitis, we may find to-day merely bronchitis, but to-morrow that the resonant note has become flat, especially over the lower part of one lung. We find also that the crepitation is finer, and this leads us to think that the bronchitis is passing into capillary bronchitis. But why are the signs more particularly marked over one lung? Because broncho-pneumonia is spreading through this lung, so that the air-cells are getting filled. With the other signs we notice an elevation of the temperature. The temperature of adults suffering from bronchitis is, to say the least, very little raised above the normal; if, therefore, in an adult patient we have signs of bronchitis and the temperature is raised above 100°, we should always suspect inflammation of the lung-substance. Of course you should bear in mind that the rise of temperature might be due, not to inflammation of the lung, but to inflammation in some other part of the body. As the pneumonia spreads, the dulness becomes more marked and the voice-sounds increase in intensity, and then we know with certainty that there is pneumonic consolidation; it is however not always easy to diagnose broncho-pneumonia, whereas in pleuro-pneumonia we do not usually find much difficulty in diagnosis. From what I have told you of the microscopical appearances we might infer that broncho-pneumonia would be a recoverable condition, and it is so, and that even in weak emphysematous lungs. Therefore, though the condition is a very serious one, do not give too gloomy a prognosis, but hold out hopes of recovery.

Let us now consider pleuro-pneumonia. In this there is a uniform consolidation right through one or more lobes, and it is therefore of this kind of pneumonia that we think when we speak of lobar pneumonia, and from its manner of onset it is also commonly spoken of as acute pneumonia. In broncho-pneumonia there is a gradual passage from bronchitis into pneumonia, but there is no sudden change, and therefore no abrupt alarm in the nervous system and no evidence of shock. On the other hand, one of the most striking features of pleuro-pneumonia is the abrupt onset. The patient is much as usual when there comes a severe rigor, with great alarm in the nervous system—the body is threatened with death. The rigor is a shrinking of the body from its communication with the outer world, and it is the forerunner of a bigger storm threatening life. Nature never breaks up a very important structure without warning. There is no surprise in Nature, and therefore people resent surprises, and rightly so. After the shock comes a severe reaction; the vessels become unduly full, so that there is a flush on the skin. Soon there is pain in the side (usually absent in broncho-pneumonia). If there is severe pain in the side, pain which hinders breathing, always suspect inflammation of the pleura. On listening to the chest at the onset of the seizure, merely harsh breathing may be heard, but in many cases a pleuritic rub soon becomes audible, and cough is often an early symptom, but it may be a day or two after the rigor before we hear definite signs of pneumonia. Commonly, however, with or without the pleuritic rub, we may detect a loss of vesicular breath-sound, and fine crepitation. In thinking of the cough of pleuro-pneumonia, remember that it is not usually very troublesome like the cough of broncho-pneumonia.

When we want to know what is going on in the lungs, we look to see what has come out of it; for that reason we take up the vessel containing the expectoration, and find that the contents consist of a watery fluid, very frothy, serum mixed with air, and we recognise that this fluid is the result of œdema of the lung. What harm is the œdema doing? From post-mortem examinations made on patients who have died in the early stage of acute pneumonia, we have been led to infer that the lung-tissues are swelled and softened by it; this has been called the stage of engorgement, the lung is exceedingly full of blood, and is œdematous and softened. The fine crepitation is due to air mixing with and pressing through this serous exudation, which has accumulated in the air-cells and capillary bronchial tubes.

The fine crepitation gradually passes away, and the breath-sounds assume a markedly tubular character; the air is evidently going into and out of small tubes; there is little or no vesicular breathing audible, merely this intense tubular sound, which shows that the sound-conducting power of the lung is much increased by the filling up of the air-cells with solid material. At this stage the percussion note is exceedingly dull, and the voice is much intensified through this solid material. What then is the condition of the lung? Its air-cells are choked up, but not with serum; the lung at this time contains very little fluid. How do we know that? When death occurs at this stage, on cutting across the lung, we find it solid and comparatively dry, the air-cells are filled with fibrin, leucocytes, and red corpuscles, and the interstitial tissue, the parenchyma, of the lung is much swelled with a similar inflammatory exudation; the pleura is swelled and there is lymph on its surface. But the bronchial tubes are not filled up like the air-cells, so that we can hear the "bronchial breathing," the sound produced by the air passing to and fro in the bronchial tubes, conducted through the consolidated tissue; similarly, "bronchophony" is the sound of the voice conducted down these patent tubes and transmitted with unusual intensity and clearness through the solid lung-tissue. A condition is apt to come on in pneumonia which prevents us from hearing the bronchial breathing and bronchophony. The exudation flows up from the air-cells along the bronchial tubes and plugs them. If the air can no longer enter the tubes, the bronchial breathing and bronchophony must of course disappear. Here let me add that you must not overlook that bronchial breathing and bronchophony may be absent in pleuro-pneumonia for another reason, because much fluid has collected between the sides of the inflamed pleura. At this period the expectoration is rust-coloured, containing fibrin, leucocytes, and coloured blood-corpuscles. This stage of pneumonia has been spoken of as that of red hepatisation of the lung.

After a time we notice in the expectoration remains of white corpuscles, charged with fat-granules, and the red corpuscles have disappeared. When death occurs at this period, the lung is no longer found in a state of red hepatisation, but is passing into a state of grey hepatisation. The solidified portion of the lung has lost much of its red colour, it contains but little blood, and the exuded corpuscles filling up the air-cells are much broken up and charged with fat-granules. These corpuscles are in appearance very similar to pus-corpuscles, and a celebrated teacher used to contend that the solid material of pneumonia was removed by suppuration. He was led to this view by the appearance of these corpuscles. It was not a useful way of regarding the resolution of pneumonic inflammation, for when we do find pus in the lungs it is under different conditions. Suppuration may, however, occur in this stage of pneumonia. Then on squeezing the consolidated lobe we see pus oozing from the whole surface, and we know then that it is in a state of grey hepatisation, with purulent infiltration.

Suppuration of this kind may break down the lung into cavities which are not due to phthisis. Purulent infiltration only comes on in pneumonia which has occurred under exceedingly asthenic conditions. We commonly meet with it in men who have drunk hard; also at the middle period of life, when the body has been under other exhausting conditions, and is much worn out. Pneumonia is in some cases so virulent that it attacks persons as if they were fatally poisoned: in a few hours they become so prostrated that they cannot raise themselves in bed.

In the individual experience of disease the question arises from day to day, How can I most usefully regard this morbid state with a view of affording relief to the patient, and of helping him to gain more health? It is, therefore, most serviceable in the study of pneumonia to group the cases into—(1) Those which recover; (2) those that prove fatal. It is necessary at the outset, when remedies have to be adopted, to recognise the natural tendency of the disease—whether the patient is likely to recover or to die.

Experience has shown that those cases are likely to prove fatal in which there has been extreme weakness before the pneumonia set in—speaking exactly, such are cases of asthenic pneumonia. Pneumonia proves fatal where there have been slow destructive changes, progressing with unerring fatality, until the inflammation ends all—a slow death spreading through the tissues before the inflammatory outbreak. In such cases of pneumonia the earlier changes may have been due to excessive drinking, or to extreme weakness owing to excessive fatigue and hurry—a maddening fatigue—until the body is killed by sleepless suffering. In others extreme weakness and excessive sensitiveness and a want of outdoor exercise prevent them withstanding cold and damp. Usually persons recover from acute pneumonia unless there has been antecedent tissue degeneration of the lung, extreme blood-poisoning, or violence.

I only see about two or three deaths a year from simple acute pneumonia where I can find by post-mortem examination no evidence of antecedent disease. Asthenic pneumonia is frequently met with secondary to Bright's disease, secondary to emphysema, secondary to fibroid or caseous changes in the lung, secondary to syphilitic fibroid thickening, or to cancerous conditions of the lung. The antecedent disease I have most commonly found after death from pneumonia has been old thickening of the lung-tissue, showing that there has been a chronic hindrance to the circulation through the lung.

When the pneumonia is secondary to blood-poisoning, to erysipelas, measles, malarial, typhoid, rheumatic, or other fever, the patient will be likely to recover, if the heart be acting healthily. Dangerous pneumonia follows violence, such as a blow on the chest, although there may be no fracture of ribs, and little evidence of external injury. Speaking of a blow leads us to think of mental shock. An accumulation of depressing influences may weaken respiration, until a few words may inflict a deeper blow than a knife. There is no doubt that words may have an immense influence when acting in conjunction with depressing circumstances. Similarly, a fright may be the cause of pneumonia, especially if associated with the influences of wet and cold.

In considering these exciting agents, we cannot but recognise that they are multiple and various; but the fact is beyond doubt, and worth repeating, that if the tissues of the body have not degenerated, the patient usually recovers. But never overlook the fact that feeling and respiration are continually working together, and that if the feeling has been extremely depressed, patients may die although they have comparatively little tissue degeneration. Hence we must direct our treatment accordingly.

LECTURE XXX.

NEW GROWTHS IN THE CHEST.

Cancer may begin in any part of the chest, but most commonly does so about the root of the lung. In cases in which there is no pressure on any particular organ, the symptoms are at the outset very obscure, and the nature of the case is not revealed until there is pressure on some vital organ. The symptoms vary according to the position of the cancer. If, for instance, it presses on the trachea near the bifurcation, the symptoms are those of tracheal stenosis; if near the apex of one lung, the disease resembles phthisis; it may begin on the pleura, and be regarded as subacute protracted pleurisy, and this may drift on to form an empyema, the cause of which may for a long time remain unrecognised; it may begin at the back of the chest, and the symptoms be mainly those of œsophageal obstruction.

Cancer at the root of the lung is by far the most common. On post-mortem examination we find a greyish lobulated mass surrounding one of the main bronchi. One bronchus is chiefly involved, but the other perhaps a little also. On cutting into the growth, much milky juice exudes, and we say at once that it is medullary carcinoma. We next want to ascertain what has been the hindrance to the processes of life. We find that the growth has caused much pressure on the bronchus, so that in this manner it has entirely occluded it; or on cutting open the tube we may find that occlusion has taken place in another way, by the growth penetrating the wall of the tube and sprouting in its interior. The patient may live for a time breathing only with the opposite lung, whilst in the affected lung the presence of the growth excites suppurative inflammation which travels from tube to tube, so that at the post-mortem examination we find that the lung consists of a mass of abscesses. The growth may spread from the root of the lung to involve the heart, especially the auricles. In other cases it may grow through the intervertebral discs, reach the spinal canal, and involve the spinal cord.

What is the suffering in these cases? At the outset the symptoms are very vague. Sooner or later the patient becomes liable to most harassing fits of coughing, followed by attacks of dyspnœa. Hæmoptysis also is very common. There is nothing to give us certainty as to the cause of the bleeding, but we suspect phthisis or aneurysm. After a time there are signs of pressure at the root of one lung, there is a drawn harsh sound on inspiration. When we hear this we say to ourselves, "Is it aneurysm, or is it cancer?"

The symptoms go on from bad to worse, but with many intermissions. Then comes a time when over one lung we can hear no breath-sound, and there is complete dulness, and complete loss of tactile vocal fremitus. There may or may not be displacement of the heart. Then the difficulty is to know whether it is pleuritic effusion, or whether there is a blocked bronchus. There is commonly a little effusion with cancer, which increases the difficulty. But it cannot be aneurysm, for no aneurysm blocks a bronchus so completely as to prevent the entrance of air.

A man was once brought to me with a pulsating tumour in the chest. I was asked, "Is it an aneurysm?" There was absolute dulness over one lung. I said, "Cancer can't produce a pulsating tumour, but I never knew an aneurysm block a bronchus like this." He had both.

A very well known medical man was brought to me by a friend, who said, "Sutton, he's come to talk to you about a cough he's got." He suffered from exceedingly severe fits of coughing, and he said to me, "Do you think it's whooping-cough?" I was suspicious that it might be cancer. Some months afterwards he had hæmoptysis, but the aspect of the case did not suggest phthisis, and I was still more suspicious of cancer. Then he became subject to recurring attacks of dyspnœa. It was, however, more than a year after I first saw him before it was possible to diagnose confidently an intrathoracic tumour.

It is often difficult to diagnose between these three—cancer, aneurysm, and phthisis—but remember that signs of extreme pressure always point to cancer.

When cancer affects the pleura it may take the form of a large tumour, filling up the pleural cavity, or it may be in small scattered masses, and the suffering varies accordingly.

The large tumour forms a medullary juicy vascular mass. There are increasing signs of pressure in the cavity of the affected pleura, and ultimately the disease may almost entirely simulate pleural effusion, so that a man with a very large experience may fall into the mistake of diagnosing cancer of the pleura as pleural effusion.

My late colleague, Dr. Herbert Davies, wrote a work on auscultation, and was a recognised authority on the subject of physical examination. He once gave a clinical lecture on a case, which he demonstrated as one of large pleural effusion on the left side. I had no fault to find with what he said, for I had previously written the following note: "There is a large pleural effusion on the left side." In went the trocar. The report was made to me: "We tapped that man, and we didn't get any fluid away." I said, "Oh, your cannula must have got blocked." "That's what we thought," was the answer, "so we put it in again, and out came a little brain-like looking substance." Then I knew what it was, medullary carcinoma of the pleura, and this was what we found at the post-mortem examination.

In smaller scattered masses cancer may excite a pleurisy which smoulders on for weeks, and this incurability is the first thing which makes us suspect cancer. It may lead to the formation of an empyema, and be treated as empyema for months, death only revealing the existence of cancer.

If the cancerous growth begins at the root of the neck, involving the glands, it may invade the apex of the lung. Such masses of cancer in the apex become caseous in the centre and soften down into cavities. These cases are easily mistaken for phthisis.

There are two or three other kinds of growth which may exercise much pressure, one being strumous change in the bronchial and mediastinal glands. These are liable to become enlarged and changed into caseous material, especially in children. They may become so large as to press on the bronchi or on the bifurcation of the trachea. I have never known such glands close the bronchus like cancer, but they may inflame, suppurate, and burst into the bronchi or trachea. There are symptoms of wasting away, with recurring and severe cough and much exudation into the tubes. Suppuration may go on for many weeks. Such a case may much resemble whooping-cough in the severe paroxysms of coughing, culminating in vomiting, but the whoop is absent. I have known the pus from a bronchial gland make its way into the pericardium and kill by suppurative pericarditis. With a similar origin I have seen severe pleurisy running on to empyema, and severe broncho-pneumonia passing on to phthisis.

Lymphadenoma within the chest may take a glandular form, the bronchial and mediastinal glands becoming enlarged and causing pressure-symptoms. On post-mortem examination the glands are found enlarged and firm, they cut and look like parsnip. They have a yellowish-grey appearance, and are much tougher than the normal glands. The growth is not limited to the gland substance, but grows into the capsule, and from the capsule grows into the surrounding structures, varying in its malignancy. In other cases the glands are not tough, though toughness is the rule. They may be exceedingly white, much swelled, and softened in the centre. Lymphadenomatous growth we find on microscopical examination to consist of lymph elements, with comparatively little connective tissue, but there is enough to make us quite sure that there is a stroma.

Another variety of lymphadenoma within the chest takes the shape of the pericardium. It seems to begin in the remains of the thymus gland, just at the top of the pericardium, and it grows down in the substance of this structure, enormously thickening it, and producing signs very liable to be mistaken for those of effusion. There is increased area of cardiac dulness, with much weakening of the heart-sounds. It is in fact impossible to distinguish the case by these signs from one of pericardial effusion, but commonly some of the glands of the neck become affected after a time, and this leads us to think of lymphadenoma. On post-mortem examination we find a yellowish-grey, tough growth, mostly made up of lymph elements, with a small quantity of stroma. It extends from the pericardium into the adjoining portion of the lung—it is evidently malignant—and thus forms a large tumour stretching across the front of the chest. There may be secondary masses elsewhere in the chest. Sooner or later in the course of such a case the spleen and the liver become enlarged. The kidneys may be correspondingly affected, in fact, glandular structures throughout the body more or less.

I will consider one other point before leaving this subject of intra-thoracic new growths. You may be asked, "What secondary growths occur in the lungs?" The most common is medullary cancer, in masses of various sizes. The smallest are of about the size of miliary tubercles, and cause a similar bronchial disturbance to tubercle, so that the case may be mistaken for one of acute tuberculosis. In other cases there may be a few larger masses in the lung. These may be entirely overlooked, their presence not being revealed by any symptom; or the symptoms to which they give rise may be quite trifling, merely harsh breathing, with occasional bronchial disturbance. Syphilitic deposits may occur in the lung, but syphilitic change here much more commonly takes the form of fibroid thickening. Hydatid is occasionally found, and also enchondromatous and fibromatous secondary growths, but the last are very uncommon. Epithelioma may occur in the lung by extension from some other organ, usually the œsophagus.

I should have mentioned that scirrhus does occur in the lung, and is liable to be mistaken for cirrhosis. In these cases there is a tough growth wandering along the connective tissue, and it is only to be distinguished from ordinary fibroid tissue by microscopical elements. Under the microscope we see a large number of cells with one, two, or more large nuclei—cells of varying shapes. Its appearance leads us to say, "This is a very progressive growth, one that will eat up all that comes in its way. It is malignant."

LECTURE XXXI.

INTRA-THORACIC ANEURYSM.

What are the usual positions of intra-thoracic aneurysm? It may occur in any part of the track of the aorta, but it is more commonly met with along the ascending and transverse portions of the arch. We must not definitely infer the place of origin from the place at which it points on the chest. An aneurysm which protrudes against the upper ribs may spring from the descending portion of the aorta. Aneurysms within the chest are commonly saccular or fusiform.

A saccular aneurysm may have a wide opening of communication with the aorta, or it may have a narrow one. The narrower the opening, the greater the chance of cure. The narrower the opening, the greater the amount of noise, and the better the sign. If then you are asked whether an aneurysm with a bruit, or an aneurysm without a bruit, is more ominous, the answer should be, an aneurysm without a bruit.

These saccular dilatations are commonly large. They involve all the coats of the artery, but notably the inner coat is extremely degenerated and atheromatous. The atheroma may extend widely down the aorta, and there may in some cases be two or three aneurysms, showing how the resisting power of the aorta is diminished. But, on the other hand, the degeneration which leads to the formation of an aneurysm may be exceedingly local. In some such cases I have had a sense of awe on looking into the body, and seeing that, while all the other organs and tissues were so exceedingly healthy, death had been caused by so limited a disease. Remember that one of the marked features of vascular degeneration is that it may be so exceedingly local.

The wall of the vessel goes on bulging more and more, and, as it does so, the coats become weaker and weaker. The outer coat presses against adjoining structures and forms adhesions, and so gets some support, but the inner coat gets softer and softer and the atheromatous material may break down. In some such cases the blood makes its way through and behind the inner coat, ripping it from the middle coat. The middle coat becomes stretched and worn, and in its turn gives way, and then the outer coat only is left to prevent rupture.

We may find on looking into the sac that it is for the most part empty, with the wall very thin in places, but more commonly upon the inner coat fibrin has coagulated, layer upon layer. The most recent layers are dark red, but the older ones are grey, their blood-pigment having been removed. But we must not overlook, firm as these layers of fibrin appear, that blood may find its way between them and soften them down again.

Now the leading idea in the treatment of aneurysm has been, and with most people still is, to bring about coagulation in the sac, so that layer after layer of blod-clot may be deposited in it, until it is completely filled. Various ways of doing this have been tried. The patient has been starved, with the idea of killing his blood and causing it to coagulate. Bleeding has been tried. Foreign bodies have been passed into the sac—needles, wires, &c. An electric current has been passed through the blood in the sac. Another way has been to apply increasing mechanical pressure, or to apply ice to the skin over the aneurysm. Various drugs have been given, such as iodide of potassium, and acetate of lead; and ergotin has been injected daily for weeks or months. I have been told in the ward, "We're curing aneurysm." "How?" I asked. "Oh, we're injecting ergotin." "Well?" "Well, the pulsation is less and the tumour is less." This was in a poor black man. He was discharged as cured. He came back again after many months. At the post-mortem examination I found the sac nearly filled with blood-clot, but the blood had made its way between the inner coat of the aneurysm and the blood-clot, and had pressed upon a weak spot until he sac had burst. The desire had been to fill the sac with blood-clot. The treatment had been perfectly successful. What is perfection but the fulfilment of an idea? It was too perfect.

Herein lies the mistake. Blood-clot does not become organised—it softens and decays. Organisation of clot may occur, but it is by means of new material coming from living tissues, and the process has nothing to do with the dead clot. Now, where can the living tissue come from to organise such a huge dead clot, lying in the worn-out sac of an aneurysm?

I once made a very curious post-mortem examination. There was a large aneurysm in the chest, and there was a hole in it. Some growth (I forget its nature) was lying pressed against the hole, so that, although the aneurysm had given way, the blood could not get out.

This gives us a hint. Get the outer coat of the sac (the adventitious coat is little diseased at first, it is the most healthy structure in the wall of an aneurysm) more and more supported; get it stronger and stronger, until the aneurysm cannot burst. Then let the sac be as clean as possible—that is to say, let it contain as little dead material, as little clot, as possible. What does it matter, then, if the blood does run in and out? It is a pathological law that whenever two structures press against one another, if the pressure is not too extreme, they tend to strengthen and support one another, and their surfaces become adherent—*cf.* thickened pleura and pericardium.

Under favourable circumstances, I regard the condition of aneurysm as a curable one. (1) The degeneration may be very limited, so that there is plenty of healthy tissue to fall back upon. (2) The outer coat becomes so strong that it cannot burst. (3) The blood runs freely in and out of the clean sac, and there is little or no obstruction to the circulation. (4) In course of time the thickened tissue of the adventitious coat contracts, and diminishes the size of the sac.

What is there to hinder such a process of cure? What is the suffering? The pressure tends to make life here unbearable. How? Commonly there is increasing pain, increasing restlessness, more and more depression of feeling.

The body's circulation is no longer in harmony with the great outer circulation. The senses cannot work freely. The pumping-out power of the lung is weak; the digestion is weak; the bowel gets weak and full; and so the circulation gets weaker and weaker.

The pain is brought about by the pressure of the aneurysm on unyielding structures, and notably on the vertebræ. In such case we get persistent pain night and day, aching pain in a very limited portion of the spine. Pain constantly in that spot night and day may be for months the only symptom. Is it spinal caries, we wonder, or is it malignant disease, or is it aneurysm? There is no rise of temperature; and after we have been impressed for months by the persistent pain, there is no marked cachexia like that of cancer, and no obvious tumour.

This pain is not always present in such cases. There was a man lying in the ward with a big pulsating mass, which I thought was an aneurysm, and the largeness of the pulsation made me suspect that the aneurysm had burst. I thought the inexperience of the clerk might prevent his getting at the history rightly, so I took the notes myself. "I was well," said the patient, "till a few weeks ago, when I was jammed by a cart against a load of bricks." But an aneurysm had eroded his vertebræ for months and months, and the accident had burst it. How obscure!

When there is aneurysm of the descending thoracic aorta, there is commonly pain—pain—pain, and no other symptoms, except the consequences of pain, for many months. An aneurysm in that situation may burst into the pleural cavity, producing the signs of a large pleural effusion, and at the post-mortem examination we find a large mass of clot, layer upon layer. Sooner or later, in the region of the pain we find dulness on percussion, and there is pulsation. As the sac pushes its way towards the surface it displaces a part of the lung, shutting out the air, so that the percussion-note becomes duller and duller. It goes on until it protrudes as a large tumour externally, and then the pain ceases. It may burst through the skin.

Aneurysm very commonly arises at the junction of the aorta with the innominate artery. Such are usually spoken of as innominate aneurysms, but the aorta is also involved. In this case the pressure is first against the nerve-structures, and neuralgia is the chief symptom—pain at the side of the neck, down the left arm, in the left shoulder—commonly called at first "rheumatic pain." Months may pass after the onset of the pain before a bruit is heard or pulsation felt.

Aneurysm is also common a little nearer the origin of the aorta. In these cases pain is most marked as the aneurysm presses towards the sternum. There is pain and shortness of breath—vague pain, with some trouble in breathing. An aneurysm in that situation interferes with the working of the aorta and with the circulation of the heart itself. Such patients may come to you complaining of pain about the apex of the heart, and you may hear nothing to account for it. There is no dilatation of the ventricle as long as the aortic valves are competent. I have seen a large aneurysm within an inch of the valves, and the left ventricle was quite normal. As long as the heart is unaffected there is always hope of cure.

Does experience in the suffering warrant hope of cure? I have known men in hospital for months get well and go back to work again. In one case the work was shovelling coals. By getting well I do not mean that the sac was full of blood-clot, but that the sac was so strong that the blood running in and out did not cause any serious inconvenience, while the heart was sufficiently strong to carry on the general circulation and to enable the man to work.

The aneurysm may spring from the transverse portion of the arch, and press on the windpipe, and probably on the left recurrent laryngeal nerve. In these cases there are spasmodic attacks of dyspnœa which nearly suffocate the patient. There is always more or less catarrh. "I always feel some phlegm clogging up just there," says the patient, putting his hand to the top of the sternum, "and as fast as I cough it up it seems to form again." On listening, we hear signs of pressure and mucous râles. The suffering in the breathing wears them out day by day. It is not the mere pressure that produces the dyspnœa; there are always some collateral acute changes which bring on the attacks.

In the still night, when the body is cold and in darkness, the breathing becomes more and more depressed. In the terrible darkness of loneliness, for loneliness is darkness, it is then that fear must come up to paralyse the right side of the heart, so that not enough blood is sent through the lungs. That is why these patients suffer from dyspnœa at night.

An aneurysm of the transverse portion of the arch of the aorta may kill by exciting broncho-pneumonia, or by bursting into the trachea. If this occurs, the patient is not, as a rule, killed suddenly and without warning; there is a little gush of blood, and then the bleeding ceases; then another, and another, and another; and finally a great rush of blood, which chokes the patient.

What are the signs of improvement brought about by strengthening the wall of the sac and diminution of its size? 1. Diminution of pain. 2. Less dyspnœa. 3. Much better nights. 4. At length, ability to move about with little or no distress.

How are we to encourage the changes which may bring about a cure? We have to get the patient over time, until the outer coat of the vessel is so strong that there is no fear of its bursting, and until the new tissue contracts and the sac gets smaller. Keep the patient much in bed for months, but do not be too strict—let him get up now and then to prevent discouragement. Let him have plenty of food. Keep his bowels working easily. Ease pain and promote sleep at night by a moderate amount of opium or morphia. An ice-bag or Leiter's tubes over the seat of pulsation will, in some cases, relieve pain and comfort the patient. Keep up in his mind a steady encouragement as to the possibility of recovery.

LECTURE XXXII.

DISEASES OF THE THROAT.

The fauces are subject to many kinds of inflammation. We often see white patches in the throat which have nothing to do with diphtheria, but result from some mild form of blood-poisoning. An instance of this is what is known as "hospital sore throat," common in nurses; we are always anxious about this for a day or two, lest it should prove to be diphtheria. The fauces are also subject to a semi-gangrenous inflammation during states of great depression.

The commonest form of throat disease is due to venous congestion. Diseases in which there is long-continued difficulty of breathing, such as vesicular emphysema of the lungs, lead to venous congestion of various organs, and one of the first regions in which this congestion is manifested is apt to be the throat. On looking into the throat, we notice that the fauces are unusually blue, unusually full of venous blood, and that the tonsils are somewhat swelled, and the uvula relaxed. The patient comes to the doctor complaining that the throat feels relaxed and dry, that there is a tickling in it, that the long uvula irritates him and makes him cough a great deal, and he asks if the uvula should be cut off. If we find that condition of throat, we should always examine the chest. In many cases we shall find that there is heart-disease, in others we shall find emphysema. We should also examine the urine, for in some cases the root of the mischief is kidney disease. As the venous congestion increases, the patient becomes liable to inflammatory attacks in the throat—tonsillitis.

There is another form of venous congestion of the throat which is very common, a condition which goes with a tendency to phthisis. In what we speak of as struma there is general weakness of circulation both of the blood and the lymph, and this weakness may be early manifested in the throat. When you look at the throat, you find that there is venous congestion, and, blending with the blueness thus caused, there is a greyish appearance due to anæmia. In such cases the tonsils are apt to become enlarged; it has long been recognised that enlarged tonsils are commonly significant of a strumous condition, of weakness in the circulation of the blood and the lymph. These conditions are very common in children and young persons, and whenever we find them we should examine the chest to see if phthisis is threatening or has already begun.

You will hear the patient say that the throat is very sensitive. As happens when there are strumous changes in the glands, these enlarged tonsils are very liable to take on acute inflammation; the tonsils become congested, swelled by serous exudation, and tend to suppurate. There are two forms of tonsillitis that we commonly meet with in connection with enlarged tonsils, suppurative tonsillitis or quinsy, and so-called follicular tonsillitis. Suppurative tonsillitis is an acute inflammation, generally more marked on one side than the other, which lasts for some days, and ends in an abscess which bursts. Follicular tonsillitis is due to an accumulation of caseous matter in the follicles of the tonsil. The caseous matter softens down, and as it does so it sets up inflammation round it. As one small mass after another is thus softened and makes its way into the throat, the inflammation recurs time after time; recurrence is most characteristic of this form of tonsillitis.

Actual destruction of tissue by strumous ulceration may appear in the throat as a prelude to laryngeal or pulmonary phthisis. I once saw a case of this kind in the out-patient department; the man had an ulcer in the throat, and the surgeon sent him over to me to ask if I thought it was strumous or syphilitic. The ulcer had an ill-defined edge, it was very deep in the middle, and had destroyed much of one tonsil; its surface was covered with large granulations. I did not think it was syphilitic, but there were no signs of phthisis. We took the man into the hospital, the ulcer continued to spread, and he died in a few months, and at the post-mortem examination we found the lungs studded with tubercle. He had died of tuberculosis, and the first manifestation had been in the throat. Sometimes a strumous ulcer, the forerunner of phthisis, may begin, not on the tonsil, but on the posterior wall of the pharynx. On the other hand, there may be gross changes in the lungs and laryngeal phthisis, and the ulceration may extend from the vocal chords along the epiglottis, and so reach the fauces, and I have known the epiglottis, the tonsils, the soft palate, and the uvula much destroyed by strumous ulceration.

Medical men often see small grey patches of exudation, and are sometimes in doubt as to the nature of these. We meet with them at the beginning of various acute diseases, such as rheumatic fever, gout, and typhoid fever, also in poisoned conditions—the so-called hospital sore-throat of which I spoke at the beginning of my lecture; and in some cases these patches are undoubtedly due to an attack of herpes in the throat. In many cases we are in doubt, we cannot decide from the appearance of the part whether what we see is the beginning of diphtheria, and we have to wait for a day or two before deciding. Sir James Paget has recorded a case of herpes in association with neuralgia of the fifth nerve; the eruption was on the face and also at the back of the throat, and the exudation in the throat looked, he says, like diphtheritic membrane. The colour of the herpetic exudation is the same as that of diphtheria; and, if we peel the patches off, they are re-formed; but there is this difference—in diphtheria the false membrane spreads, and commonly spreads widely, whilst in herpes there is no such spreading, the exudation is limited to the site of the original vesicles. I have seen such recurring herpetic membrane also in herpes of the mucous membrane of the lips, but there was no spreading at the edges.

I will describe to you a typical case of herpes of the throat which occurred in one of my own colleagues. As he was driving down to the hospital he felt very chilly. "I thought," he said, "I was going to have an attack of rheumatic fever." He shivered, his limbs ached, his temperature rose high, he had severe headache. The next day his throat was sore, and we were much struck by its appearance. There was extreme venous congestion, giving the throat a bluish colour, differing markedly from the fiery red congestion of diphtheria; on this congested surface there were red papules; he was somewhat delirious. At the end of the second day there were raised white patches, and the next day these patches were more distinct and almost vesicular; he had laryngeal stridor, and wanted me to send for the surgeon to open his windpipe. It was evident that the mucous membrane of the larynx was affected; the obstruction to the breathing led to venous congestion of the kidneys, and he had a fourth of albumen in the urine, there was much delirium. It was with difficulty that I could persuade myself that it was not diphtheria. The next day the temperature fell to normal, the laryngeal stridor had ceased, there was no longer albumen in the urine, and he was soon well.

Erysipelas sometimes affects the throat. Whenever there is inflammation of the neck from any cause, examine the throat from time to time; the inflammation may spread through the connective tissue of the neck, and while our attention is riveted to the outside, œdema of the glottis many come on and kill the patient. In the first case of erysipelas of the throat I saw, I was struck by the extreme œdema. If there is erysipelas of the skin, and an œdematous inflammation of the throat comes on, we know that this is erysipelas of the throat. In cases of erysipelas of the skin we should frequently examine the throat, and should listen for laryngeal stridor, so that we may be ready to perform tracheotomy if necessary; in such cases this operation is most useful.

I will speak to you here of one other cause of œdema of the throat. The surgeons call it phlegmonous inflammation. That means an "ugly indescribable kind of inflammation." I have only seen it in people who have been great drinkers, who have bad blood, and are very rotten. They get this inflammation of the throat, laryngeal stridor comes on, and they die of obstruction to the breathing. Your only chance in these cases is to treat them boldly, and try to get out the poisonous products. Salivate them, bleed them, purge them—

in fact, you must try to kill the patient before the disease does. There is one more morbid condition of the throat of which I must speak. Whenever the body has been much weakened, and there has been catarrhal inflammation of certain mucous membranes, a favourable medium is afforded for the growth of the spores and mycelium of a peculiar fungus, and we get the condition known as thrush. Diphtheritic inflammation never travels down the œsophagus; its progress is always sharply limited at the lower end of the pharynx. If therefore on the post-mortem table we see a quantity, it may be a very large quantity, of yellowish material on the mucous membrane of the pharynx and œsophagus, ending abruptly at the opening of the stomach, we know at once that it cannot be due to diphtheria. If we examine the deposit under the microscope we see granular matter, the débris of epithelial cells, mixed with spores and mycelium. It is thrush.

This condition may come on in connection with catarrhal inflammation of the mucous membranes in all cases in which there is extreme asthenia. It is common towards the end of diabetes and phthisis, it is common in very old people, and in exceedingly weakly infants. When do we know that diabetes is going to kill? There is a flush on the cheeks, the lips are red and raw, the tongue looks like a piece of raw beef, and on looking at the throat we see white spots on the fauces. "It looks like curdled milk," we say, "the patient has been taking milk." But it is not milk, for it is not quite easy to detach it from the mucous membrane, and on microscopical examination we see that it is a fungous growth. The actual growth does not extend down the windpipe, but its neighbourhood is apt to set up catarrhal inflammation of the windpipe, extending to the bronchial tubes, and this bronchitis may be enough to kill an exhausted patient. We must therefore be on the watch for thrush in all chronic exhausting diseases attended by extreme wasting and asthenia, for in such thrush is often the immediate cause of death. I have known thrush set up diarrhœa, and keep it up for weeks. I once had a very instructive case—a child with diarrhœa, which all the usual methods failed to stop. At last my attention was called to some white patches in the throat. Under the microscope I recognised the spores and mycelium of thrush. I gave a few grains of borax daily, and the child rapidly got well.

It is most encouraging to see how the fungus disappears when we give borax internally—grs. xx, three or four times a day. Give the borax internally, for the fungus grows in the submucous tissue, and you must "rat it out."

LECTURE XXXIII.

STRICTURE OF THE ŒSOPHAGUS.

Stricture, in the rigid sense of the term, means an extreme narrowing of a canal from the contraction of fibroid material which has formed in the wall. This condition is rare in the œsophagus. When we do find it, it is usually the result of swallowing some irritant poison, a strong acid or alkali; this sets up a sub-acute inflammation, fibroid tissue is formed in the wall of the œsophagus, and in the course of many months this material undergoes thickening and contraction, so that the passage is almost closed. I have never met with stricture of the œsophagus as a result of syphilis. Syphilis sometimes leads to stricture in the trachea, but it does not cause stricture in the œsophagus, nor anywhere in the gastro-intestinal tract, excepting the rectum.

Ordinarily, when we speak of stricture of the œsophagus we mean something different from this. The obstruction exists during life only; we find it clinically, but we cannot find it on the post-mortem table. The patient is unable to get the food down into the stomach, and when the medical man passes a bougie, it goes down to a certain point and there it meets with much obstruction, but often with gentle force it is possible to overcome the obstruction, and the bougie passes on into the stomach. At the post-mortem examination we find that there is no narrowing, but that the wall of the œsophagus over a large area is much destroyed, and we see that, owing to the destruction, this part of the œsophageal wall has been paralysed.

In these cases there are two kinds of obstruction, the one active, the other passive. The active obstruction is due to a condition of irritable weakness of the œsophagus immediately above the ulcerated part, so that a spasmodic contraction of the part is excited by the contact of food or bougie. The passive obstruction is due to the paralysis of the œsophageal wall at the seat of ulceration. And let me add that the greatest obstruction we meet with is passive obstruction—it is impracticable. If a thing offers no resistance, we do not know how to deal with it.

What is commonly known as stricture of the œsophagus is malignant ulceration of a portion of the œsophageal tube. It may occur in any part of it, but is more common in the middle or lower part. It is usually either epithelioma, or medullary carcinoma, whilst scirrhus is rare. I prefer, at present, to use the old terms in speaking of morbid growths. In epithelioma we find a mass of ulceration which has entirely destroyed the mucous membrane over a large area. The edge of the ulcer is a raised, thickened, grey mass, and outside the edge there are small nodules in the mucous membrane which have not yet broken down into ulcers. Medullary carcinoma begins more to the outside of the œsophagus, and extends inwards, breaking down into an ulcer as it reaches the surface of the mucous membrane. It is a thick, lobulated, very juicy and vascular growth. I have a few times seen an ulcer with a clean well-defined edge, and I have in very rare cases seen a similar ulcer on the mucous membrane of the intestine; these cases were perhaps syphilitic. We very commonly find that the ulceration has invaded adjoining structures—most commonly it extends into the trachea, but it may be one of the bronchi, or the pleura, or the apex of one of the lungs.

We will now turn to consider the suffering, which is much the same whatever the nature of the ulceration. How the early suffering begins I cannot tell, for the disease is usually far advanced before it comes under the notice of a medical man. A time comes when the patient notices that he does not get solid food down as well as usual. He comes to the doctor and tells him that he is unable to get food down, that it comes up immediately; that he is getting very weak, and falling off very much in weight. If we listen carefully to him we notice that he does not say that he vomits, but that the food comes up; and we know from his manner of stating his symptoms that he is not suffering from stomach disease. There is no conscious effort in bringing up the food, and in that respect it differs from vomiting; and there is little or no pain. He says he feels the food go down to a certain point, and it either comes up immediately, or it stays for a minute or two, and then returns into the mouth. From these symptoms we infer that there is œsophageal obstruction.

As the disease progresses, there comes an extreme difficulty in getting even liquids down. But we notice this curious fact: some days the patient may be able to get solid food down into the stomach, and some days a bougie can be passed and other days it cannot. We have not to deal with a mere mechanical narrowing, and the condition of the parts varies from time to time.

The stomach is cut off from the outer world, and the body is awfully depressed. It thirsts and hungers, and this not merely for food and drink: there are hunger and thirst after life. As starvation comes on we have symptoms of wasting and dryness; there is wasting of the muscular system, and increasing asthenia; the belly is sunken, the fæces are hard and dry, leading to much constipation, the urine is scanty, and the skin is dry. The parts of the body which owe their roundness to the presence of fat in the cellular tissues, such as the cheeks, the temples, and the orbital region, become sunken, for the fat of the body is almost entirely consumed. But it has often been noticed that life can be much extended if the body can get water. The patient will live for weeks, taking hardly anything besides water: the utmost failure does not come until he is absolutely unable to swallow fluids. The pulse is extremely small, for the heart is starved also. But it is astonishing how little these patients complain; they are too depressed to complain.

It has been thought advisable in these cases, when the patient is obviously starving to death, to endeavour to pass a bougie forcibly down the œsophagus, and in this way to overcome the obstruction. Of course we can pass a bougie if we push hard enough, but what will happen then? The result of such treatment has been a more rapid extension of the ulceration into adjacent parts. The operation, I was assured, was a success, but the patient died! I have not gathered much encouragement from that line of treatment. All we can do in these cases is to try and relieve their suffering a little, and we shall hardly do that by forcibly passing a bougie, and increasing the risk of the extension of ulceration into the trachea, the lung, or the pleura. What is wanted is to get more blood through the seats of spasm and of paralysis—energy comes from blood. Sometimes I have given the sufferers a little opium to ease them through their misery, and they have swallowed better for weeks afterwards. Sometimes a little alcohol has eased the circulation, so that the patient has been able to swallow better for a time.

When the ulceration has extended into the trachea or bronchi, the patient becomes liable to attacks of violent and severe coughing, coming on after attempts to swallow; these are brought about by the passage of food into the air-passages. This condition usually leads in a short time to death from pneumonia. In other cases perforation of the pleura by the ulceration sets up a fatal pleurisy. The commonest mode of death is however by slow starvation.

Before leaving this subject I will refer briefly to another cause of difficulty in swallowing, and of perforation of the œsophagus. An aneurysm of the descending thoracic aorta, lying against the œsophagus, may perforate its wall and lead to death from hæmorrhage. Do not be misled by finding that the hæmorrhage is not large at first. There may be a little bleeding from time to time, and this may continue for days and even weeks before the final great gush of blood which kills the patient. An aneurysm bursting through the skin or into any of the internal cavities commonly bleeds in this manner. I remember a case of aneurysm in a small pulmonary cavity resulting from the fracture of a rib; the patient spat blood again and again for several days, until at last there was a great gush of blood which choked him.

LECTURE XXXIV.

INDIGESTION.

What do we understand by indigestion? Indigestion is a disturbance of feeling with an exceedingly intricate cause. Indigestion means that there is an undue hindrance to the passage of food into blood—of pabulum into man. The stomach empties itself, but the man is not refreshed. Pounds of food are eaten, but the man remains miserable.

Are there several varieties of indigestion? Some observers have made several, according to particular symptoms, and we may make any number. If we wish to make a specialty of stomach we can make a number of varieties according to special features. Dyspepsia itself is only a symptom manifesting that digestion is interfered with, and rendered disturbing to the mind.

All strong processes go on easily, comfortably, and unconsciously. A healthy man does not feel that he has a stomach, and as soon as a man comes to feel that it, depresses him. Hence with indigestion there is usually mental depression, accompanied by a sense of weariness; there is fulness, pain, or other distress in the region of the stomach; there may be vomiting; often there is palpitation of the heart, or aching about the præcordium. Such symptoms may last for some hours after a meal, giving evidence of slowness of digestion. When the digestion is weak there is excessive accumulation of gas in the stomach, and as the gas is eructated it brings up with it some of the acid constituents of the digestive process, and acidity may be the trouble complained of. Patients often attribute their trouble to the acidity. In the healthy process of digestion the stomach moves the food to and fro. There is a kind of churning process to mix its contents, and promote the onward flow and absorption of the pabulum. It is, no doubt, a rhythmical action. When the stomach is much dilated, some of the wave may be seen transmitted externally. When that healthy movement is interfered with by over-distension with gas or other cause, vomiting commonly comes on ; undigested food is vomited. If there be much persistent irritation of the stomach, catarrh may supervene, and then there is vomiting of acidulated mucus and undigested food. If the indigestion be very protracted various forms of fermentation may lead to decomposition of the food in the stomach. The vomited matter in such cases is a frothy, yeasty-looking, decomposing substance; the process of decomposition may go on till sulphuretted hydrogen is formed, and then the vomit smells like rotten eggs.

We will now take a more physiological view of indigestion. For healthy working of the stomach there must be healthy structure, and there must be a sufficient supply of healthy blood ; there must also be a sufficient supply of nervous energy, for this, as disease in nerves reveals, is a great co-ordinator, keeping the organs of the body working harmoniously. One great function of vis nervosa is to keep the organs and tissues of the body harmoniously working with the travelling fluids, and in relation to digestion it enables the stomach to be the servant of all the organs and tissues of the body, and to serve the purposes of mind. To enable the mind to be a power, supposedly the greatest power, the stomach supplies our nature with the products of the world. Therefore, men who have very active minds commonly do and should eat well, and they may be large eaters. When there is excessive thinking and great wakefulness the stomach craves for food—there is much sinking sensation in the epigastrium, and patients have often said to me, "Oh, doctor, I must take a biscuit up to bed with me." Experience has taught me that when there is excessive action of mind, wakefulness, and delirium, much food is indispensable.

I used to be in the habit of saying that people ought to "eat properly," that they should only eat every three or four hours, that they should not eat between meals, they should eat and live by rule; but year by year many poor creatures complained dreadfully of their stomachs, and of sinking sensations, and they became so miserable their days were almost unbearable. The big bell of Westminster tolled hourly on, that is, properly, and their times for eating were regulated accordingly. But as I grew older I looked at the animals, and they were eating and eating, and evidently whenever they could. I learned at last to thank Heaven that I, and others, had been made as animals, and with use of reason. Let people eat when it is necessary, let them take food when they want it. The stomach, proceeding with healthy digestion, is an immense encouragement to the human mind.

Medical practitioners often hear patients complain that they cannot take this or that article of food, it distresses them so—it may be salmon, shellfish, or even mutton or fowl. In these patients it is evident that there is some condition of sensibility which, when brought in connection with processes of digestion and influences of food, discourages and distresses much. It is usual, therefore, for the practitioner to direct the patient to avoid eating such food, and only to take so and so. In doing that he hopes to encourage, and endeavours to satisfy—his plausible manners no doubt conducing to his success; but it must be admitted that such a proceeding requires great judgment. For the patient credits the practitioner with doing that which is most conducive to his (the patient's) welfare, strength and progress. It must reasonably be recognised that digestion means going for "newness"; taking food is a new state of things, for "refreshment"; and all change in the body must be attended by some alteration in sensibility. The brain, the heart, the lungs, the bowels, and all the organs, respond to the actions of the stomach.

That is evident from the sufferings of the patients. In health there is unity and evenness in the correspondence and interchangeability, consequently there is not distress but comfort, not intimidation but trustfulness.

Now, in reference to dietary, we have to consider how the wants of the fluids, tissues, and organs for strength are to be satisfied; and the practitioner will not learn this if his thought is narrowed to hard-and-fast decisions, and if he thinks he knows and does not inquire what the individual's needs are—if he does not feel his way into unknown regions, and let Nature guide him.

It is very instructive to notice that children and old persons suffer little or not at all from indigestion, and incline to eat anything that is nice. The child takes the world on trust, and the aged mind has had enough of dicta. There is much truth in the old saying, that a man is either a fool or a physician at forty.

I have witnessed enough, as regards indigestion, to convince me that it is better some distress should be suffered in giving the means to enable the stomach and other organs to benefit by change of diet, rather than keep the individual from various foods, which are the vehicles carrying influences from sea, earth, and heaven, to enable us to learn what is man's relation on this earth.

For healthy digestion there must be healthy rhythmical action of the stomach, and for that proceeding it must not get over-distended. Then there must also be healthy tissue in the wall of the stomach, muscle that can contract rhythmically, capillaries that can let out flowing serum, for healthy secretion and digestion. There must also be healthy epithelium for absorption, and healthy lymphatics for transmission; there must be a free passage onwards to the other organs; healthy action of the lungs to carry on the venous circulation, for the lymphatics empty themselves into the venous system; healthy action in the liver, to work up the food; healthy action in the bowels and other excretory organs, to maintain freshness in blood and structures. There must be healthy expenditure of energy, for living beings take in health in proportion as they give out. A person who stays indoors too much, and leads a miserable life, loses appetitite. For healthy digestion there must also be healthy desire; and for that purpose there must be sufficient change in food and in associations in general. "Change is in all things sweet," and the physiologist has come to the conclusion that change of food much assists digestion. These remarks are testified to in a remarkable manner in the sufferings of melancholics. They wish to sit still without speaking, and to be reduced to one or two articles of food; they suffer dreadfully in their stomachs, their ideas are so set. The world is going beautifully on and all things are progressing, but not to them. Their loneliness would lead them to keep still and starve to death; they are weary of the hard and narrow decisions which have reduced poor self to desolation, and shut out a lovely world.

LECTURE XXXV.

CATARRH OF THE STOMACH.

One of the most common diseases of the stomach is catarrh. People will tell you that they have a "cold in the stomach." Just as some persons when exposed to cold get sore throat, and others nasal catarrh, so others get catarrh of the stomach, leading to distress in the stomach and vomiting of mucus.

When we speak of catarrhal inflammation, we mean a more severe morbid condition. The mucous membrane is exceedingly swelled, especially along the greater curvature, where the blood gravitates most. There is more or less œdema, giving the membrane a watery appearance; it glistens somewhat, and is much congested. The congestion is mainly venous, so that the membrane has a purple colour. If on post-mortem examination we find a bright red arterio-capillary congestion, we immediately think of poisoning, especially of poisoning with arsenic.

This leads me to give you a caution about the stomach. You must not overlook that it is usual after death from various diseases to find the stomach much congested. This congestion is proportional to the amount of blood in the body. If therefore there has been a great deal of blood in the body, and there has been very rapid death, we generally find extreme congestion of the stomach, and we must not hastily conclude that it is due to poison. If, however, we find no evidence of organic decay to account for death, we have to consider the possibility of poisoning, of strangulation, or other violence. You will easily get your guide from the appearance of the stomach, as well as from the history. The venous congestion due to "dying" is always most marked along the greater curvature, and on holding the stomach up to the light, the veins can be seen filled with blood; whereas in cases of irritant poisoning there is arterio-capillary congestion, widely spread over the whole surface of the stomach. If a patient is suddenly taken ill, vomits, and dies, and at the post-mortem examination we find no evidence of organic decay, remember that death may be due, not to poison, but to epilepsy.

Where congestion has been extreme, we often find hæmorrhages into the mucous membrane. Scattered here and there we see very small particles of black deposit. On lifting one of these we see that it is a small blood-clot, lying in a little pit. Such superficial ulceration commonly occurs in connection with extreme forms of catarrhal inflammation of the stomach. The condition is known as punctate ulceration of the stomach, or, if the ulcers are a little larger, guttate ulceration. Such a condition is commonly seen after death from heart-disease. It is owing to the venous congestion, catarrhal inflammation, and superficial ulceration of the stomach, that persons with heart-disease suffer so much in their stomachs. Do not overlook this fact; for patients who have heart-disease go to the doctor for relief of the stomach distress, whilst others go complaining of heart when their suffering arises from stomach disease. In the latter cases, as the stomach becomes excessively charged with gas and digestion is difficult, the action of the diaphragm is doubtless hindered and the heart's rhythm interfered with.

What do we see under the microscope in cases of catarrhal inflammation of the stomach? There is astounding morbid change. It is indeed difficult to get a healthy stomach for microscopic examination from the post-mortem room, owing to the changes which commonly come on in the process of dying. The epithelium is extensively swelled, so that we see what appears to be a hyaline albuminous substance, and much of it has been destroyed. The capillary walls are also swelled, and so is the connective tissue between the capillaries—swelled and charged with leucocytes and red corpuscles. The epithelium of the peptic tubes is clouded and charged with albuminous matter.

Where there has been long-standing venous congestion and repeated catarrhal inflammation of the stomach, as in heart-disease, pulmonary emphysema, and hepatic cirrhosis, we not only find the changes just described, but in course of time the connective tissue of the stomach has become much thickened, and has invaded the muscular wall. There is much deposit of pigment, which at once attracts our attention, and leads us to say, "There has been chronic obstruction to the circulation through this stomach." This condition was formerly called cirrhosis of the stomach.

Various morbid conditions may give rise to catarrh and catarrhal inflammation of the stomach. Anæmia and cachexia of various kinds, especially the cachexia of Bright's disease; venous congestion, from heart, liver, lung, brain, or other organic failure; exposure to cold; morbid growths in or about the stomach, notably cancer; various blood-poisons; and very commonly it results from abuse of alcohol.

Catarrh of the stomach is evidenced by hyperæsthesia, some uncomfortable weariness, aching, or other distressed feeling in the epigastric region, more or less constant, but increased by taking food. Bear in mind that catarrh of the stomach is always most marked along the most dependent part, the greater curvature, and that food, immediately it is taken, must come in contact with that part of the stomach. Owing to the weakening of the muscular coat by venous congestion, the patient is much troubled with wind. The pain in the stomach is relieved by vomiting, but not entirely removed. The removal of the contact between the food and the stomach immediately lessens the pain, but, since the swelling of the mucous membrane persists, the pain is not entirely abated. The vomit is a watery fluid, mixed with mucus and food. If the inflammation is very severe, vomiting may occur repeatedly in the absence of food from the stomach. The vomit is then simply a mucoid substance. It is the vomiting of mucus, with or without food, which reveals that there is catarrh. There is no feverish condition. There may be streaks of blood in the vomited matter; and in its most extreme form, when patients are dying with it, they vomit mucus mixed with blood, looking like beef-tea or coffee-grounds. It is the black vomit of the dying.

Remember that an important sign of failing circulation may be repeated and severe vomiting. Rapid and severe failure of circulation or breathing, producing extreme venous congestion of the stomach, may lead to frequent and severe vomiting, which may be the only symptom complained of. If ever you have to deal with a case of anæmia in which uncontrollable vomiting comes on, direct your mind to failing heart.

A number of people will come to you complaining of vomiting. "Whatever I take comes up," says the patient. You find no organic disease, but it may be impossible to stop the vomiting until you get the patient to lie up for a while. He has been exhausted. In these cases there is a morbid condition of the stomach which requires further investigation. There is great disturbance of the stomach with more or less catarrh, and it appears to be due to what people speak of as "worries"—extreme mental anxiety. In some such cases the pain and vomiting may be exceedingly severe, and I have known vomiting of blood, and I could not, from antecedent and subsequent course, attribute it to ulcer. So I would have you consider that, under extreme mental depression and harass, the stomach, like the face and other surfaces, may become congested and undergo catarrhal inflammation, and the vessels become so full that hæmorrhage may result.

Bearing on that statement, experience has shown that there may be vomiting of blood, perhaps frequently repeated, so that the hæmorrhage has been attributed to ulcer of the stomach, until the post-mortem examination revealed that there was no ulcer of the stomach, but morbid change in the brain—a tumour. It may also occur in connection with some epileptic or insane condition.

Not uncommonly catarrh of the stomach offers some difficulty in distinguishing it from simple ulcer of the stomach, particularly where there is anæmia. Anæmic young women are liable both to catarrh and to simple ulcer of the stomach. Sometimes we have to ask, "Has the morbid condition passed beyond catarrhal inflammation into simple ulceration of the stomach?" There is anæmia, there is pain after food, there is vomiting, and this relieves the pain—such symptoms are common both to catarrh of the stomach and to simple ulcer—how then are we to tell one from the other? This will be considered in my next lecture.

LECTURE XXXVI.

SIMPLE ULCER OF THE STOMACH.

How are we to distinguish between simple ulcer and catarrh of the stomach? Simple ulcer of the stomach, in by far the majority of cases, occurs along the lesser curvature, and therefore the pain does not come on immediately after food, but at a time varying from a quarter of hour to as much as two hours after food is taken. Only in the rare cases in which ulcer is along the greater curvature will the pain come on immediately, as in catarrh. In simple ulcer of the stomach there is not usually much swelling of the mucous membrane, therefore, the uneasiness is not persistent as with catarrh of the stomach. When they have vomited the patients are free from pain; when they lie down in bed they are free from pain; whereas with catarrh the membrane is more or less swelled, and there is continual uneasiness. Owing to the seat of the morbid change, the pain of ulcer is felt not only in front, but passes through from the lower end of the sternum towards the angle of the right scapula. The more lumpy the food the worse the pain, and therefore we should give a liquid diet. Further, with simple ulcer there is hæmatemesis, whereas with gastric catarrh it is rarely met with. Lastly, an important sign is, that with simple ulcer, if the patients be kept in bed with liquid food, they speedily get relief to their symptoms—more quickly than they do in catarrh. I have seen a large, simple ulcer, like a "rodent ulcer," in the stomach; the patient was a young woman, and she suffered dreadfully with pain night and day until she died. Such pain is rare. In simple ulcer, pain is rarely felt when the patient is lying quietly in bed at night, a marked difference from what we see in cases of cancer.

The relief to the pain experienced on lying down leads us to think that it is caused by the pressure of the liver and the diaphragm on the stomach.

Simple ulceration of the stomach is best grouped as it is naturally into acute and chronic. By chronic cases we mean simple ulcer of the stomach, recurring and recurring over years.

The simple ulcers in their acute form are usually small, about the size of a threepenny-piece or sixpence, but they may be larger. They look as if a piece of mucous membrane had been chiselled out—there is a definite clean-cut edge, without any marked thickening. They are usually funnel-shaped, tapering down to the peritoneum, and hence are liable to cause perforation and rapid collapse. If, therefore, the symptoms are similar to what I have described, we should always keep the patient lying down, so that if the ulcer be near the peritoneum, there may be time for inflammatory action to be set up, and for the wall of the stomach to become adherent to some neighbouring organ (since the ulcer is usually at the lesser curvature, this organ is most commonly the liver), and so perforation be prevented. It is notorious that the symptoms, in cases of this kind, may be readily overlooked. Hence, I repeat, if there is any reason to suspect ulcer of the stomach, put the patient at rest in bed. The experience of different hospitals would supply sad evidence of that failure in diagnosis. I remember the case of a young man who was attacked with collapse from perforation as he was leaving this hospital, and I found that simple ulcer of the stomach had caused his death.

When perforation takes place, patients are suddenly seized with severe pain, extreme restlessness, laboured breathing, cold sweats, and the pulse becomes exceedingly small. A case of that kind reminded me very much of the collapse of cholera, and I could not make any distinction except in the origin. The symptoms of perforation, if not so severe, may be followed by peritonitis; food escaping into the peritoneal cavity excites peritonitis. We must not forget that the peritonitis results in much lymph being effused, which may surround and localize the escaped food, and the patient may ultimately recover. I remember making a post-mortem examination where a simple ulcer had perforated, and there was an old buried-up abscess containing concrete pus over the scar of the ulcer.

Simple chronic ulcer of the stomach may cause a considerable amount of thickening about the stomach wall, and I have known this mistaken for cancer. In this case the bearing of the history was not sufficiently recognised; the stomach irritation had lasted for years. In chronic ulcer of the stomach there is usually a much larger area of mucous membrane destroyed than in simple ulcer. It is a recurring ulceration, and therefore the ulcer is much larger, and is surrounded by much scar-like fibroid thickening. The ulceration frequently opens into the vessel, and therefore recurring hæmatemesis is common, this and the other symptoms of gastric ulcer being repeated over many years. We are led to infer that in the fibroid tissue which forms around these chronic ulcers the vessels are very weak, for they are buried up in tough hindering material, like that in any scar. When the general circulation is weakened the fibroid substance is liable to be swelled by serous exudation from the weak vessels, and take on acute inflammation.

When asked, "What is the cause of simple ulcer of the stomach?" say, "I don't know." It occurs with anæmic conditions, therefore it is most common in young women who are much indoors; but it occurs also in males, and in connection with several cachectic conditions, such as Bright's disease, and others. It has been suggested that it is due to thrombosis in the stomach. I can only tell you that I have often taken up a stomach and seen the veins more or less plugged, without a trace of simple ulcer; but it might be replied that there is no similarity in such conditions, that with simple ulcer there is a localized death in the blood, and, owing to the anæmia, also much failure of nervous energy, tending to lead to death in the tissues. There is much to be considered about the solar and cœliac plexuses, in relation to simple ulcer of the stomach, and to nervous distress in the stomach region.

LECTURE XXXVII.

CANCER OF THE STOMACH AND PYLORIC OBSTRUCTION.

Cancer may attack any area of the stomach, but it is most commonly met with along the lesser curvature, lying between the lesser curvature and the liver. These cases are extremely obscure at the outset, for the growth lies out of reach, behind the lower end of the sternum. The patient's suffering, increasing failure of strength, loss of flesh, and distress persisting day and night, evidently denote severe disease. The weary uneasiness in the epigastric region makes it exceedingly difficult to get sleep. Muscle continues to waste, and healthy inclinanations and feelings are replaced by the utmost gloom. Food is often vomited, and becomes more and more mixed with quantities of mucus and frequently with blood. We are then able to say that there is some persistent irritation of the stomach, keeping up catarrh, and that this is associated with failure of constitutional power and nutrition. It resists all treatment, therefore we think that it is malignant.

If we keep our minds on realities, we can keep clearness of mind. I say that here, for a searching question comes up in these cases, and about this stage of the proceedings. Are the symptoms indicative of cancerous, or of simple ulceration of the stomach?—which further means, Can we go on hoping for recovery? In simple ulcer, if patients rest in bed, they usually lose their pain, therefore they are not so much disturbed at night, and they do not lose flesh to the same extent. "Time," said the old English Judge Hale, "is the wisest thing under heaven;" therefore go on hoping, until the symptoms are beyond question.

After a while we feel increasing resistance in the epigastric region, a sense of abnormal solidity, and at last an irregular, nodular, deep-seated growth is felt; then we know it is a cancerous growth extending through the wall of the stomach. As it becomes more nodular we have no doubt; and the general symptoms make it more evident by their increasing severity.

Cancer originating in the duodenum may grow up into the stomach. In these cases intestinal symptoms may predominate, or the bile-duct may be blocked, causing jaundice, or there may be pressure on the portal vein, leading to ascites, and with these conditions symptoms of stricture of the pylorus may be blended. Cancer originating in the œsophagus may grow down into the stomach, and in this case the predominant symptoms will be those of stricture of the œsophagus.

In all these cases the growth is usually medullary carcinoma. We find an irregular, fungous, ulcerated surface, which has grown freely in the stomach wall, causing much destruction. The growth is very juicy and very vascular. Such a growth may begin in the wall of the stomach, and extend in towards the cavity, and out towards the liver. In such cases the pyloric orifice becomes more or less closed, leading to vomiting, dilatation of the stomach, and rapid wasting. In these cases, in which the pylorus is strictured by medullary carcinoma, the dilatation of the stomach never becomes so great as in the more typical cases of stricture of the pylorus, for the patient is too ill to take much food, and progress towards death is much more rapid. Usually a tumour is sooner or later felt through the abdominal wall.

In other cases there is no vascular growth, but the middle coat of the pylorus becomes enormously thickened, and grows in and closes the orifice more or less completely. In cases of this class we may find evidence indicating that the morbid change has resulted from abuse of alcohol. The pylorus forms a small tumour which may sometimes be felt through the abdominal wall between the gall-bladder and the kidney. When we cut into the pylorus we notice the glistening appearance of the middle coat, and we are astonished at the excessive quantity of the greyish-yellow circular muscular fibres. The peritoneum and the mucous membrane may also be a little thickened, but there is no ulceration. We notice also the great size of the stomach, and that the whole muscular coat has undergone some compensatory hypertrophy. The degree of dilatation depends on the amount of food that has been taken. The stomach may be empty, but is usually filled with a sour decomposing mass. When we think of this horrid stuff in the living stomach we feel an irresistible desire to wash it out.

In the most chronic cases of pyloric obstruction, in which the disease may last for years, with intermissions, the obstruction is caused by adhesions. In one case I saw there had been gall-stones which had caused inflammation about the gall-bladder, and adhesions had formed distorting the pyloric end of the stomach, so that when the stomach contracted there was a twisting valvular action, closing the outlet. In other cases contraction and thickening at the pyloric end of the stomach may be the remains of simple ulceration. I used not to know that the symptoms of pyloric obstruction might recur from year to year, but experience has taught me not to give an unfavourable prognosis. If the muscle gets more power the food will pass on—the obstruction largely depends on weakness of the muscle.

What is the kind of suffering when stricture of the pylorus is coming on? An obscure indigestion is usually the first symptom—fulness and discomfort after food—which resists treatment, and there is not any very definite condition to account for it on; heart disease, neither renal disease nor phthisis, nor other organic failure. After awhile vomiting supervenes, but what rivets the attention more is the increasing sense of fulness, and the increasing accumulation of food in the stomach. As time goes on the patient vomits more frequently undigested food, and we have then to ask, how it is that the food is not digested and passed onwards. The hindrance is not due to any accidental condition, for the difficulty continues week after week; the loss of flesh reveals that there is something seriously hindering the passage of the food into the general nutrition. We next notice that the vomited matter is charged with much mucus, and this affords evidence that there is some persistent irritation of the mucous membrane, which leads us to think that there may be a morbid growth.

We further notice that the vomited matter smells like vinegar and looks yeasty, indications of acetous fermentation in the stomach, and there is fungous growth in it in the form of sarcinæ. After awhile the retained food commonly decomposes more, and sulphuretted hydrogen is formed, so that the vomit smells of rotten eggs. While this is going on, the belly gets more and more sunken, the eyes are also sunken —the flesh and fat of the body diminish. The fæces are harder and increasingly scanty, as the food passes less and less into the intestine. Water in sufficient quantities cannot get beyond the stomach, and the urine becomes scanty and loaded with lithates. The skin also is dry, for the whole body is being dried up. In this manner the patient becomes progressively weaker, and dies from starvation.

In the early period of the disease the vomiting may be very frequent, but as the stomach becomes stretched we notice that enormous quantities are vomited at intervals of perhaps two or three days. The vomiting is in some cases more distressing to the patient at the early stage. A patient with extreme stricture of the pylorus may go on for weeks without vomiting, and all the while the food is being impacted in the stomach, layer upon layer, until the stomach is almost filled—excepting a small passage in the middle of the impacted mass through which liquids might pass on into the duodenum—so that the stomach becomes unable, from excessive distension, to contract, even for weeks, while impaction is going on. Such cases I have seen.

The failure in these cases is not merely due to the stricture. As food accumulates and decomposes in the stomach, a quantity of gas is formed, which still more excessively stretches the stomach, and increasingly tends to paralyse it. It is important to recognise that, for if we can get rid of these gases and the decomposing food, it is astonishing to see how much the patient improves. Formerly disinfectants were resorted to—hyposulphites, creosote, charcoal, and others; but more recently the stomach has been washed out, to get rid of the decomposing food and gases. By so removing the over-distension the blood is enabled to pass through the vessels more freely, and store the muscular tissue with energy for contractions, and so the stomach is enabled to get the food through the strictured pyloric orifice.

But perhaps your patient will say to you, "What's the use of my living to have my stomach washed out every day?" I know I should feel like that myself. I had one patient with pyloric obstruction whose mouth I turned into a stomach. He chewed beef all day without swallowing it, and got so much better that he was able to leave the hospital.

The stomach is subject to simple atrophy. The wall wastes away, and in rare cases this condition is the immediate cause of death. Owing to the wasting of the muscle the stomach becomes greatly dilated, and the food lodges there, so that the patient dies with symptoms resembling those of pyloric obstruction—there is, indeed, a passive obstruction, and the cases are clinically exceedingly difficult to distinguish from those of stricture of the pylorus. A more common and lesser degree of atrophy is associated with wasting diseases, such as phthisis, diabetes, &c. Think of the possibility of the existence of atrophy of the stomach and intestines in cases of severe chronic constipation.

In many forms of stomach disease, but especially in those last described, wind accumulates in the stomach, and of this the patient must be relieved. Gastric distension and discomfort have an immense influence on breathing. This deficiency in breathing leads to the irritability so noticeable in persons suffering from flatulence. There is a degree of unbearableness in suffering from flatulence which we must relieve, or it will kill the patient. Suicide from this cause is not uncommon.

LECTURE XXXVIII.

ULCERATION OF THE INTESTINES.

You may be asked to enumerate the different forms of ulceration of the intestine. They are—

1. Simple ulcer of the duodenum.
2. Ulceration of the duodenum, which is said to occur after burns.
3. Ulceration arising from poisoning.
4. Typhoid ulceration.
5. Tubercular ulceration.
6. Dysenteric ulceration.
7. The ulceration which gives rise to perityphlitis.
8. Ulceration occurring in the stage of reaction of cholera and diarrhœa.
9. The "black ulceration" of scurvy.
10. Ulceration which is liable to come on in protracted venous congestion of the bowel.
11. Syphilitic ulceration.
12. Cancerous ulceration.
13. Ulceration arising from the continuous pressure of a mass of hardened fæces in the rectum.

Typhoid fever, dysentery, and typhlitis are considered in separate lectures, so I need only touch upon them here. The clinical features and post-mortem appearances of cancerous and syphilitic disease of the bowel have more dependence on stricture than on ulceration, so that they are better considered under the head of intestinal obstruction. I have touched on the subject of catarrhal ulceration in the lecture on cholera and diarrhœa. The more important varieties remaining for description are then, simple ulcer of the duodenum, ulceration from poisoning, which however I considered at some length in the lecture on typhoid fever, tubercular ulceration, and the "black ulceration" of scurvy.

Simple ulcer of the duodenum is like simple ulcer of the stomach, and its cause is similarly obscure. It is situated near the common duct, and gives rise to symptoms like those caused by the passage of gall-stones, but without jaundice. Jaundice, however, might come on from catarrh extending into the duct. There are frequent attacks of severe pain coming on after meals and relieved by vomiting, and there is tenderness over the region of the duodenum. With such symptoms in a young subject, think of ulcer of the duodenum. These ulcers may perforate the gut.

Superficial ulceration of the duodenum is said to occur in cases of burns of the skin in which the patient has survived two or three weeks. We have naturally little opportunity of verifying this statement by post-mortem examination.

As I may not again have an opportunity let me here say a few words on other diseases of the upper part of the intestine. The duodenum is very little subject to disease. Catarrh is not uncommon, and usually arises in connection with dyspepsia. By extension of the catarrh to the bile-duct, jaundice arises. The duodenum may be invaded by cancer which has begun in the pylorus, the gall-bladder, the pancreas, the kidney, &c. The most common of these is cancer of the head of the pancreas, growing into the duodenum and blocking the common bile-duct, so that a most intense jaundice is produced.

Primary disease of the jejunum is practically unknown. Typhoid or tubercular ulceration may extend up to it from below, and catarrh may attack it in progress up or down. This rarity of primary disease is a very instructive fact, for the jejunum with its valvulæ conniventes is a storehouse of food. Look at it after a meal and you will find it covered with a rich creamy substance. Were it diseased, nutrition would rapidly suffer, and frequent primary disease of this part of the bowel would be incompatible with the continued existence of the species.

Phthisical or tubercular ulceration of the intestine is not always associated with phthisical changes in the lungs, though such association is the rule. In rare cases of strumous bone or gland disease we may find some yellow caseous-looking tubercle in the mucous membrane of the ileum, and a few superficial ulcers. In these cases there has usually been diarrhœa, and diarrhœa has probably been the immediate cause of death.

In an ordinary case we find the Peyer's patches and solitary glands the seat of more extensive ulceration; there is very little characteristic deposit, but large irregular granulations occupy the floor of the ulcers; they are not very deep, but are commonly more or less pigmented. When we see these appearances we at once ask, "Are they tubercular?"

In most cases you will find no tubercle in or around the ulcer, but in some cases you may find enough caseous deposit to make you sure it is tubercular. But when you look at the peritoneum covering the floor, that is, from the peritoneal surface, you will see grey miliary tubercles, about the size of a pin's head. Herein lies the chief distinction between the typhoid and the tubercular ulcers. In books we are usually told that we may distinguish between them by the fact that the typhoid ulcer has its greatest length in the direction of the length of the bowel, whilst the tubercular ulcer extends across the gut. This is one of the things which the writers of books copy from one another year after year without referring to the facts. I have frequently seen tubercular ulcers extending along, and typhoid ulcers extending around, the bowel. If you are asked to give the distinctive features of tubercular ulcers, say—

1. We find miliary tubercles on looking at the peritoneal surface of the ulcer.

2. There are large irregular granulations over the whole surface of the ulcer, reminding us in appearance of those we see on strumous ulcers of the skin.

3. The ulceration is associated with phthisical changes in the lungs or elsewhere.

4. There is none of the deposit characteristic of typhoid fever in the neighbourhood of any of the ulcers.

In passing, let me say that the grey, semi-translucent, gelatinous deposit in the Peyer's patches and solitary glands of the ileum is absolutely characteristic of typhoid fever. In malarial dysentery there is a deposit in the solitary glands of the colon and rectum which very much resembles it, and which undergoes ulceration and sloughing like the deposit of typhoid fever, so that if a small portion of the rectum were cut out in the acute stage of malarious dysentery, and put beside a similar piece of intestine from the lower end of the ileum in a case of typhoid fever, I could not myself distinguish between them. But whereas in typhoid fever the morbid process is always most extreme in the immediate neighbourhood of the ileo-cæcal valve, and above, rather than below (for severe affection of the large intestine is rare in typhoid fever), in dysentery the great stress of the inflammation falls on the lower part of the rectum, while the inflammation never extends above the ileo-cæcal valve. I am speaking of malarious dysentery, which is the only form of dysentery in which we meet with this grey deposit.

There are one or two clinical distinguishing features between typhoid and tubercular ulcers of the intestine. It is not at all uncommon for a typhoid ulcer to perforate the bowel, but perforation is exceedingly rare in tubercular ulceration; hence peritonitis, common in the former, is rare in the latter. Further, in typhoid ulceration, severe hæmorrhage is common, and hæmorrhage is often the direct cause of death; in tubercular ulceration severe hæmorrhage is rare, and I cannot recall a single fatal case.

The ulceration of the intestine we meet with in cases of diarrhœa and cholera is superficial. We find numerous small superficial ulcers, lying in a dark-red congested granular mucous membrane.

What are the characters of the ulceration of the intestine that occurs in persons suffering from scurvy? We cannot see the early changes in the intestine, but we can watch them in the mucous membrane of the mouth. The first thing we notice is a small blood extravasation on the gum. In twenty-four hours we notice in the centre of the hæmorrhage a brownish-yellow discoloration, and this rapidly becomes a slough. Now, in the intestine, both small and large, we find a number of black hæmorrhagic areas, each with a brownish-yellow slough in the centre—these are known as the black ulcers of scurvy. Their appearance at once makes us think of scurvy, and we look for similar ulcers in the mouth, and for subcutaneous and inter-muscular hæmorrhages.

Tertiary syphilitic disease of the intestine, other than syphilitic fibrous stricture of the rectum, is exceedingly rare. When we meet with it we find ulcers very similar to tertiary ulcers of the skin, they have clean-cut edges, and, if they have lasted some time, we find whitish areas of fibroid thickening around them.

If in the ileum we find one or two ulcers only, well-marked ulcers with definite edges, and no other morbid change, we think of poisoning, and of poisoning dating from two or three weeks back. My experience of these cases has been as follows: the patient has taken some corrosive poison, most commonly it has been a strong alkali, and at first did not seem to have suffered severely. When we looked into the mouth we found little or no destruction of the mucous membrane, and congratulated ourselves on the slight nature of the case. For ten days or a fortnight all seemed to be going on well, and then the patient got rapidly weaker, the pulse became weaker and weaker, until death occurred. On post-mortem examination the only morbid change we found was such ulceration as I have described.

LECTURE XXXIX.

OBSTRUCTION OF THE BOWEL.

Let us define what we mean by obstruction of the bowel. We mean simply that the bowel cannot get the fæces along. Now this may obviously depend on one of two conditions; either there is an abnormal resistance interposed, too powerful for the intestinal muscle to overcome; or else the intestinal muscle has by some cause been so weakened that it is unable to overcome the normal resistances to the passage of the fæces; or these two conditions may be combined in varying degrees. Now, except in certain cases in which surgical interference is possible and advisable, as in hernia, merely to think of the interposed resistance is useless in practice; what is wanted is to gain more peristaltic energy along the intestine, and to find out when and how that energy is expended unavailingly.

It will perhaps be best to speak first of the slighter degree of failure of intestinal action known as constipation. In many persons constipation appears to be an inherited condition, and is unchanged for years. The bowels may act only twice a week, or even less frequently, but I have not found that it has led to any harm. It is commonly associated in such subjects with nervous affections, and is common where there is a tendency to phthisis, insanity, or diabetes. I find it difficult to speak of any relation between constipation and improper feeding. It has not come within my experience.

A common condition, taking away the energy of the bowel, is failure of circulation, and therefore constipation is common in anæmia. Failure of the circulation is commonly due to starvation, and hence in various starving morbid conditions there is constipation, and, may be, constipation to such a degree that we cannot get the fæces out. The circulation of the bowel may be much weakened in heart-disease, and constipation be the result. We sometimes see it in obstruction to the venous circulation in emphysema of the lungs. The interference with the venous circulation may be within the abdomen; in cirrhosis of the liver we commonly get attacks of diarrhœa alternating with constipation, and a similar symptom is seen in cases of malignant growth obstructing the portal vein. Constipation may be the sequel of inflammatory changes in the bowel, which have damaged its circulatory power, and hence it is not uncommon after typhoid fever and cholera.

Failure of nervous energy is a most important cause of constipation. The simplest instance of this is the constipation commonly complained of by men who persistently overwork their brains. But we meet with the most serious cases of this where there is spinal cord disease. I remember one case of a young woman who had been lying on her back for months; the medical attendant had to take a spoon and scoop the fæces, piece by piece, out of the rectum; they had got packed and hard, and the bowel was utterly unable to pass them on. Whenever nervous energy has been much exhausted, notably in hysteria and other nervous conditions connected with insanity, we are never suprised if there is great difficulty in passing the fæces, and we may have to remove them by mechanical means. Again, where there has been a great discharge of nervous energy, after childbirth for instance, the bowel is liable to be much weakened, and becomes greatly distended and tympanitic, and the patient is unable to pass the fæces; a few nights' sleep and a little mechanical aid remove the trouble.

There is a condition known as atony of the bowel. This is characterised by persistent accumulation of fæces which the bowel cannot expel. Masses of fæces may be felt through the abdominal wall during life. After death, when we open the bowel from one end to the other, there is not a small passage, but an excessively large one—the bowel is by paralysis loaded with fæces and dilated. The wall of the bowel is usually very thin. The nature of this condition is obscure.

There is another condition of which I may as well speak here—it is too much regularity of the bowel. There are people who pride themselves on "never missing a day." By-and-by this becomes exceedingly harassing. They miss a day, and it makes them exceedingly nervous, for the last twenty years they have gone every morning before breakfast. It is a terrible thing for such people to become irregular. "Oh doctor," they say, "if you could only get my bowels open I should be quite well." The idea has become a tyranny. Dr. Barlow once told me that he had been called to see a man whose bowels had not been opened for six weeks. He said to me, "Sutton, why shouldn't a man go for six weeks without any actions of the bowels." And I answered, "Why not, if he's comfortable?"

There is a class of cases in which there has been some peritonitis, and this has weakened the bowel, or else has left much adhesion of the coils. Under favourable circumstances the bowel has sufficient energy to deal with the difficulty, but when from any cause the circulation is weakened, the symptoms of acute obstruction supervene. I remember a case in which soon after the operation of ovariotomy the patient got increasing distension of the belly and other signs of obstruction and died. There was extensive adhesion, but no sign of mechanical obstruction; evidently the obstruction was due to paralysis of the bowel.

The remaining conditions which give rise to symptoms of intestinal obstruction may be conveniently considered under three heads. 1. Cases in which there is softening and paralysis of the muscle of the bowel, as in peritonitis. 2. Mechanical blockage, leading to the muscle being exceedingly stretched until it is paralysed. 3. Obstruction due to cancerous or other growth.

Obstruction of the bowel may result because the muscular coat of the intestine is softened by the œdema of inflammation. Sometimes in such a case the surgeon is called upon to open the abdomen, for there is very evidently some irremediable obstruction. Then the question arises, "Is it certain that there is a mechanical obstruction, or is the obstruction due to paralysis of the intestine consequent on the muscular coat being softened by peritonitis?" I repeat, that peritonitis softens by œdema the muscular coat of the intestine, and doubtless the nervous structures also, until the bowel is unable to pass the fæces onwards. Such patients may die with symptoms like those of mechanical obstruction. A further similarity is brought about by the fact that unrelieved cases of mechanical obstruction usually go on to peritonitis.

What are the varieties of acute mechanical obstruction. The commonest of all is hernia, inguinal, femoral, or the rarer varieties. Secondly, there are cases with essentially the same causation, but the seat of constriction is well within the abdomen; the gut may be strangulated under a band of peritoneal adhesion, or may slip into some hole in the mesentery, the result of abnormal fœtal development. These cases may be called internal hernia. Thirdly, there is intussusception, occurring chiefly in children, and with a peculiar symptom, differentiating it from the other forms of acute obstruction. In perityphlitis the symptoms are in part those of acute obstruction, but I shall deal with this condition in another lecture. Lastly, I must mention the extremely rare cases in which acute obstruction is produced by the impaction of a foreign body, most commonly at the ileo-cæcal valve; it may be something swallowed or it may be a gall-stone which has ulcerated through from the gall-bladder into the duodenum.

What happens when a portion of the gut is strangulated? The venous circulation in the strangulated portion becomes extremely obstructed, it turns purple in colour, and œdema and swelling set in. Then there is a copious albuminous exudation, increasing swelling and density. Since this swelling occurs in a confined space, the vessels of the tissue are pressed on more and more, until the arterial and capillary circulation are cut off; then ulceration may begin in the mucous membrane and lead to perforation, or the tissue may slough in mass.

The intestine above the impacted and strangulated portion becomes extremely distended from accumulation of liquid fæces and of gas. As the accumulation takes place there is an increasing tendency to the production of peritonitis, starting from the seat of stricture, and extending up along the bowel.

The peritonitis and the stretching tend more and more to paralyse the bowel. At the post-mortem examination we find the strangulated piece of bowel purple, and perhaps sloughing, whilst above the strangulation the bowel is excessively distended and its peritoneal coat is inflamed; there may be general peritonitis, and the gut may have been so soft that it has given way.

Before we go further let us consider the pathology of acute obstruction of the bowel. What are the effects of bowel obstruction? The greatest danger lies in the increasing tension within the belly. As the pressure increases it tends to depress the breathing, and as the breathing becomes more and more depressed there is increasing difficulty with the gaseous elements in the intestine above the obstruction. When a piece of bowel is strangulated the first warning symptom is pain. Colic sets in, and becomes more and more violent. It is that increasing violence of the colic, making the patient bend and twist about, due to excessive peristalsis, that marks acute obstruction. There is much less colic in peritonitis. As the pain increases vomiting sets in. At first it is only the food and drink that were last swallowed, but in a little while the vomit smells of fæces, and then we have no doubt that we have to deal with obstruction of the bowel. If the obstruction persists the patient soon comes to vomit simply liquid fæces. Then the belly becomes increasingly distended, the cheeks sink in, the eyeballs become depressed, the hands blue. The breathing is labouring, and the temperature tends to fall below normal.

We must not measure the danger by the number of days the obstruction has lasted, nor by the pain, but by the condition of the pulse and breathing. As the tension becomes dangerously great the pulse becomes smaller and harder, and the breathing harsher, the veins in the neck stand out, and the lips become blue. Perhaps for a time the vomiting may give relief, but sooner or later, if the obstruction remains, the patient tends to pass into collapse. The pulse now becomes soft, and if anything in the way of an operation is to be done, it must be done speedily. For many hours no urine may be passed, for the water is drained out of the body by the excessive vomiting. Gradually the circulation fails and the patient dies.

I spoke above of the distension of the abdomen, but you must remember that if the obstruction is high in the jejunum, or in the duodenum, there may be very little distension. In these cases the vomiting comes on early, and there is less pain. The lower the obstruction, the greater the distension of the abdomen ; where the process is a slow one, and the block is at the lower end of the bowel, as in cancer of the rectum, the distension is often extreme. I use the word often, for there are cases of cancer of the rectum in which there is no abdominal distension, as I shall explain more fully later. In the diagnosis of acute cases, remember that little distension and early onset of vomiting, with comparatively little pain, point to obstruction high up ; whilst when the constipation is absolute for several days before vomiting comes on, when the distension is very great, and the pain very severe, the obstruction is at the lower end of the bowel.

Let us now ask a very important question. Why is it that the piece of bowel became strangulated when and where it did ? The bands found are old structures, and had been present for months, at least, before the obstruction. It seems obvious that the healthy circulating energy of the bowel had been such that it kept its safe position and its rhythmical peristaltic action ; but from the fact that it became impacted and strangulated it might reasonably be inferred that the circulating energy had been failing prior to the impaction. We have often noticed in examining bodies that, as the circulation had failed the bowel had become irregularly distended or contracted, and we can understand that, given a possible seat of impaction, such irregular contractions might lead to its occurrence.

What we have to do in all these cases is to consider how healthier rhythmical peristaltic action can be restored. This attempt gives a rational basis for the opium treatment. It is of no use to drive hard by aperients, for this simply results in the body being taken to the dead-house. We have to consider how the respiration and heart's action can be aided, to promote a better circulation through the bowel. Opium is given in these cases because it rests the patient.

If we can relieve pain, the breathing becomes freer, and the venous circulation is more promoted, thereby enabling the arterial circulation to supply the muscle with more energy, and thus the nervous energy of the bowel increases, and we give the healthy part of the bowel a chance to get the impacted part out of the difficulty. So that the great question is, how to get more blood and energy through the intestine. The great cause of obstruction coming on is failure of circulation, and that especially in the structures weakened by old inflammatory or other morbid change. I have found such remains.

The history of some of these patients reveals that they have had minor warning attacks of obstruction before the fatal seizure. So much have I found this to be the case that I endeavour never to under-estimate the importance of such warning attacks; they show the onset of the weakness of circulation which leads to the obstruction. I may mention the case of a schoolmaster who was in the hospital. He had partial paralysis of the hands and feet from spinal cord disease. One day he was seized with severe colicky pains, and died rapidly of obstruction of the bowel. On post-mortem examination I found that his intestine had been strangulated under an old band. Now his general circulation had been failing for months before the strangulation occurred.

Before I leave the subject of acute obstruction let me give you two more facts. First, one other cause of symptoms such as I have just described, is what is known as volvulus. In this condition a portion of the bowel, most commonly the sigmoid flexure, becomes twisted on itself, so that it is as it were strangulated by itself. Round the root of the twist another portion of the bowel may be twined—in some cases even knots are formed in the intestine. The venous circulation in the bowel is interfered with, and the subsequent changes and the symptoms are similar to those of strangulation under a band. The cause of these twists is somewhat obscure, but there is some evidence to show that they also are the result of failing circulation in the bowel. The second point about which I wished to speak is, that symptoms exceedingly like those of acute intestinal obstruction are apt to come on in cases of lead poisoning.

Intussusception forms in a sense a link between acute and chronic obstruction, for its course is less rapid than the former, and more rapid than the latter, By intussusception we mean that a portion of the intestine has been squeezed into the the portion immediately below it, turned outside-in, invaginated. It is always an upper portion passing into a lower. As a longer portion passes in, the venous circulation becomes increasingly difficult, and the invaginated portion turns of a purple colour. Swelling and exudation follow, just as in strangulation, but more slowly. The swelled and congested mucous membrane tends to bleed and bleed, and this, while it retards the moment when the circulation must entirely cease in the invaginated bowel, gives rise also to the characteristic symptom of intussusception—melæna.

If then in a child—the condition is not uncommon in children, but is very rare in adults—there are symptoms of obstruction, colic, vomiting, and abdominal distension, and with these symptoms there is repeated hæmorrhage from the bowel, the case is probably one of intussusception. There may be two other things to guide us in recognising this condition; sometimes we can feel a long tumour, corresponding to the intussusception, through the wall of the abdomen; this varies in position from time to time, and sometimes we cannot feel it at all, though it may have been quite distinct a short time previous. This is because the muscular contraction of the intussusceptum varies, and if it is lax we may be quite unable to feel it. Again, where the belly is much distended it may be quite impossible to feel any distinct tumour. The second thing I spoke of is, that sometimes the invaginated bowel may be pressed down as far as the rectum, and may be felt and seen in the rectum as a dark purple mass.

The patient may die of collapse, or the bowel may be withdrawn by a natural effort, and the child rapidly recover. In some cases the invaginated portion sloughs off. How do we know that it sloughs off? Because it is discharged externally and we see it. The contiguous portions of the peritoneum and the other structures of the intestinal wall just above the invaginated bowel become adherent, and as the lymph becomes organised the bowel is healed—united again.

In this as in other forms of intestinal obstruction it is the tension within the belly that kills the patient, and the aim of the medical man should be to lessen that tension until the invaginated bowel sloughs off, or is withdrawn. In some cases we may succeed in pushing up the invaginated bowel by injections of water, air, or oil; in other cases the abdomen has been opened by the surgeon, the invaginated bowel has been pulled out with the fingers, and the patient has recovered.

In these cases also it has been instructive to find that there have been warning attacks before the major attack, and I have been led to think that weakness of circulation was behind that accident also, a slowly creeping on weakness. Another fact may guide you to consider failing circulation as the condition which leads to intussusception. For intussusception, it may be in several parts of the bowel, is often found after death, and as there were no symptoms of intestinal trouble during life, and as these portions of the bowel are not congested, it is obvious that they were formed only as the circulation through the bowel was at the point of cessation. It may be reasonably inferred that when some portions of the intestinal wall have lost their power, a peristaltic wave of energy coming along may not be sufficiently transmitted by the powerless, more dead portions, and the energy of the wave, not being transmitted in its accustomed direction, carries the moving piece of bowel into that which refuses to act.

What are the conditions that give rise to chronic obstruction of the bowel? Cancer is the most common, whilst syphilis is rather rare. In some cases there has been some inflammation in the pelvis, the result of perimetritis, and much fibroid material has been left, and this contracting has led to stricture of the rectum. There may be an annular fibroid thickening surrounding the bowel, the remains of ulceration, whether tubercular, typhoid, or due to irritant poisoning. All these are very rare, stricture after typhoid ulceration being exceedingly so. In some cases carcinoma may begin about the bladder, or uterus, and extend to the rectum.

Sometimes the cause of obstruction is outside the bowel altogether, for instance, pregnancy may occur in a retroverted uterus, or an ovarian cystoma may be situated in Douglas' pouch—as these grow we have the symptoms of chronic obstruction produced by pressure on the rectum.

Cancer of the bowel occurs in two forms. In the first, annular cancer, there is a ring of malignant growth about an inch, or half an inch, in diameter—so small that it may be difficult to find even on post-mortem examination. This small annular cancerous growth is most common in the ileum, and as a rule we can feel no tumour during life. At the post-mortem examination we find a grey juicy vascular mass completely surrounding a portion of the intestine. When we cut into it we see that the mucous membrane has been destroyed by cancerous ulceration; just above the growth the gut is somewhat dilated, but not much. We may or may not find cancer elsewhere. These cases are very obscure, and extend over months. The growth usually causes irritation, and sets up enteritis, leading to diarrhœa, and thus obstruction and abdominal distension are usually absent. Even if the obstruction is very great, if the growth is high up, in the jejunum for instance, there will be little distension, but vomiting will come on early. The usual history of these cases is this. The patient is between thirty and fifty years of age. He has repeated attacks of colicky pain in the belly, griping pain, night and day, for months and months. There is loss of flesh, and loss of healthy colour, with extreme mental depression. There is diarrhœa, and a quantity of mucus mixed with blood is passsd from the bowel, thus affording evidence of protracted catarrh. You ask what it is that is keeping up the catarrh; putting all the symptoms together you are certain that there is severe organic disease, and you think that it is probably cancer.

In the other form of cancer of the bowel there is a mass of cancer sufficiently large to be felt through the abdominal wall, if it is not out of reach from the front, being situate in the rectum. It is usually greyish and very vascular, showing a tendency to hæmorrhage. The peritoneum around is somewhat inflamed and thickened. Within is an ulceration which has destroyed much of the mucous membrane and invaded the muscle, destroying it likewise. Above and below the seat of growth the mucous membrane is swelled and thickened by catarrhal exudation. The bowel above the growth is distended and dilated. Such a growth is usually found in the sigmoid flexure or in the rectum. In some cases of cancer of the rectum the growth is much buried in fat, so that at the post-mortem examination we need to go very carefully to work to make sure that there is a malignant growth at all.

What is the suffering in these cases? Pain is of course a prominent symptom; if the growth is low in the rectum there is frequent desire to go to stool, and pain before, during, and after defæcation. Often some mucus and blood are passed with the stools. If there is no diarrhœa, which is the prevailing symptom in some cases, there is increasing constipation and distension due to accumulated fæces and gases, until the bowel becomes enormously dilated. As the tension and irritation increase the breathing becomes very weak, and the circulation and oxidation in the cerebro-spinal centres is much diminished, so that there is great depression and restlessness; anæmia comes on and there is great irritability, which you must endeavour to relieve. As the distension reaches the duodenum and stomach vomiting comes on, but it is a late symptom, and as the obstruction increases there is often vomiting, not only of gas and of food, but at last of fæces. Here let me warn you that you must not, because there is vomiting of fæces, think that there is certainly obstruction. Such vomiting simply means regurgitant action of the bowel, and I have known it occur in Bright's disease.

Here let me say a few words about colotomy. In some cases it undoubtedly makes the suffering more bearable, for it relieves the tension in the belly, but it is not always advisable. There was a case in which Sir James Paget has advised colotomy at first, and had then changed his mind, and I was called in to give an opinion. I found that the patient was a man who, apart from the disease, had been through a great deal of nervous strain. I advised against the operation, for I thought that the sudden change in the condition of the gut might kill him very rapidly. "Will the operation cure him?" "No, nothing can do that." "Will it relieve this awful desire to go to stool every few minutes?" "If he lives more than a few days after the operation, and I don't much think he will, it may relieve that, but in his case I don't feel quite sure that even then it will relieve his suffering." "Then we won't have the operation done. What do you advise us to do?" "Let him have a bottle of brandy a day." "Oh," they said, "he's drinking a bottle and a half already."

These patients can consume enormous quantities of alcohol and opium without their heads becoming affected.

In some cases of cancer of the rectum and sigmoid flexure, owing to the catarrh, diarrhœa is the main feature. The patient may go to the doctor to complain of diarrhœa—there may be more or less griping, frequent desire for stool, liquid evacuations frequently mixed with blood and mucus—the diarrhœa may be so excessive as to produce a sunken belly, and yet the bowel may be nearly blocked with cancerous growth. Therefore, it is incumbent in all cases of very obstinate diarrhœa to examine the rectum for cancer, and occasionally, especially in middle-aged persons, you will find a mass of growth.

Syphilitic stricture may be situated as high as the ileum, but is most common in the rectum. What is to guide us in distinguishing between cancerous and syphilitic stricture of the rectum? Both produce a large, thick, hard mass in the wall of the rectum. Both commonly have much fat mixed with the morbid growth, so that the tumour is of a considerable size; both cause ulceration and destruction of the mucous membrane; both contract and constrict the rectum, leading to fatal obstruction; in both on cutting into the mass on the post-mortem table we find it tough and fibrous. How do we distinguish between them? 1. Syphilis commonly occurs in younger subjects. This is of course no great help, for syphilis may occur late, whilst cancer of the rectum may kill before the age of thirty. 2. The course is more chronic, there is less pain and wasting, and a less cachectic appearance. 3. Besides the gummous deposit, and the fibroid change at the seat of stricture, there is often fibroid change in the mucous membrane below the stricture, and this fibroid change may even extend through the anus to the skin of the buttock. Then we are sure it is syphilitic. 4. Often there is a well-marked tertiary ulcer, like those found on the skin, below the stricture. 5. Commonly there is well-marked syphilitic change in other organs.

Let me before leaving this subject sum up for you the chief dangers of stricture of the rectum. 1. Peritonitis may come on at any time, and prove fatal. 2. There is a risk of suppuration in the neighbourhood of the stricture. 3. There may be ulceration due to enteritis just above the stricture, and this may lead to perforation. 4. In these cases collapse may set in rapidly and unexpectedly. All these chances should be taken into consideration when judging on the advisability of colotomy.

A word in conclusion about ulceration of the rectum. It is a story of a case which was a lesson to me. I was going round the wards one day, and they pointed out a patient to me, saying, "We can't get that girl's bowels open." It appeared to be a case of simple fæcal accumulation, and I told them to give her an enema of gruel and turpentine. The next time I came round I asked how she was, and they said she was dead. "Dead!" "Yes, we gave her the enema of gruel and turpentine as you told us, and she died in a few hours." I made the post-mortem examination, and found the gruel and turpentine floating about in the peritoneal cavity. There was an ulcer in the rectum, nearly through, and the force of the injection had caused perforation. This case will teach you, as it taught me, that injections in cases of intestinal obstruction must be given with extreme care and slowness.

LECTURE XL.

PERITYPHLITIS.

I HAVE to-day to speak of an important morbid condition of the intestine, an inflammation of the tissues in the neighbourhood of the cæcum, and hence called perityphilitis. It is known by various other names—typhlitis, and disease of the cæcal appendix. This disease is characterised by very definite symptoms; and in the large majority of fatal cases we find, at the post-mortem examination, that there has been ulceration of the cæcal appendix to start the morbid process, and therefore we conclude that in the many cases that recover the same structure is at fault.

The cause of the ulceration of the appendix is obscure. At post-mortem examinations, small, hard, dark bodies are not uncommonly found in the diseased appendix, and for many years it was commonly supposed that these were the stones of fruit. But if we cut into such concretions we find that they merely consist of fæcal matter; and though foreign bodies such as fruit-stones may undoubtedly set up perityphlitis, such cases are very rare in comparison with those in which the offending body is formed in the intestine itself.

We next have to ask how it is that the intestine has allowed the concretion to form, and we have no sufficient answer to give. We know that typhlitis is not uncommonly associated with, or followed by, phthisis, and it is not unreasonable to suppose that there may have been a kind of chronic catarrh, whether simple or strumous, leading to paralysis of the muscle of the appendix, and that thus stagnation takes place in its interior, and a concretion may easily form. Once formed, it acts as a continuous irritant until ulceration is set up.

In some cases the ulceration goes on quite insidiously until perforation occurs; there is fæcal extravasation, and rapidly fatal purulent peritonitis. But it is impossible during life to determine the cause of the peritonitis. In all cases of peritonitis in which, at the post-mortem examination, you are in doubt as to the cause, do not forget to examine the cæcal appendix.

More commonly an inflammatory irritation spreads from the base of the ulcer, causing what may be called conservative peritonitis. This, by causing adhesion of the contiguous coils to the floor of the ulcer, prevents the fæcal extravasation from taking place. If there is inflammation set up in the connective tissue around the cæcum (perityphlitis), this spreads to the muscle of the cæcum, paralyses it, so that fæces accumulate in it, and a passive obstruction is set up.

In some cases the irritation is sufficiently intense to lead to suppuration, and an abscess forms in the connective tissue in the neighbourhood of the bowel; or it may be that there is a localised peritoneal suppuration, cut off from the rest of the peritoneal cavity by adhesions. The abscess thus formed may burst into the bowel, and the cavity close up spontaneously; or it may point towards the skin, and be opened by the surgeon; or it may travel in both directions, and a fæcal fistula result. Lastly, it may perforate the tissues which have shut it off from the general peritoneal cavity, and the patient may die of a general peritonitis.

In the majority of cases, however, perityphlitis consists simply in ulceration of the appendix, attended by some non-suppurative inflammation of the connective tissue around the cæcum, some local peritonitis, with inflammation of the muscle of the cæcum, and more or less complete intestinal obstruction. After the symptoms due to these conditions have lasted for a few days, and have perhaps been exceedingly threatening, the patient commonly recovers.

What is the suffering in perityphlitis? As a rule, the first indication that there is anything amiss is that the patient complains of pain in the right iliac region, and on examination we find that this region is tender. The pain and tenderness may only last for a few hours, and then disappear, to return in a few days; and there may be two or three warning attacks of this kind.

When the attack is definite and developed, there is severe pain, beginning about the umbilicus, as in acute obstruction, pain making the patient double up, and twist and turn; he dreads to have his abdomen touched; there is usually constipation, and may be severe and repeated vomiting. At this stage it may be difficult to determine whether the case is one of acute obstruction, of acute general peritonitis, or of perityphlitis.

On careful examination, however, we usually find that the right iliac region is more tender than other parts of the abdomen, and often, when we put the hand there, the patient will tell us, "I have had something there for some weeks." He finds it difficult to keep his right leg down, because when the iliacus is relaxed the inflamed region is less tense. On percussion we find that the right iliac region is dull. Then we know at once that there is not ordinary gas-filled intestines beneath the abdominal wall. There is lymph effused around the intestine, and an accumulation of fæces within.

A condition tending towards collapse now comes on. The pulse is small, the eyes are sunken, the temperature is below normal, and a cold sweat breaks out on the skin.

Your aim must be to limit the peritonitis, and to guide the circulation into reaction. As reaction occurs the face becomes flushed, the temperature rises, the breathing is quickened; there is some delirium; and there may be signs of diffused peritonitis, as a distended belly and diffused tenderness.

Often the condition of the patient looks hopeless. You can hardly feel his pulse, and his face is sunken and blue; but having seen a great many of these cases now, I never give them up till I know that they are dead, for they commonly recover. They have a very good chance; for the disease usually occurs in young subjects with much rallying power and no tissue degeneration.

Now it is most important to avoid hurrying patients in this condition with stimulants. You must give time for the peritonitis to seal up the fæces, so that general peritonitis shall not occur. Just keep the patient resting; give him light food, and a little alcohol; and, above all, do not forget to put in a word of encouragement. Remember that disease of the belly is a terribly depressing thing, and that depression tends to kill. If you simply and quietly say to the patient, "You will pull through it," you will help him a great deal.

In about three or four days the pulse becomes stronger, the face is less sunken, and the breathing becomes freer. "Will he escape without suppuration?" is the question we now anxiously ask. This depends on the intensity of the irritation. If suppuration is going to occur the tumour remains, the temperature keeps up, and week after week the symptoms of local suppuration continue. Do not in these cases be unduly anxious, for in these, also, the patient commonly recovers, after the abscess has discharged, either externally or into the gut.

Does persistence of the tumour always mean suppuration? No. I recently had a case in the wards. The boy remained ill week after week; there was some fever, there was constipation, and a tender swelling in the right iliac region. Week after week there was no change, and no local redness. It seemed probable to me that there was no suppuration, but that the inflammation had paralysed the muscle of the cæcum, and that thus had come about an accumulation of fæces in the paralysed bowel; I thought that this was keeping up irritation, inflammation, and fever. I therefore told my house-physician to give him a gentle aperient. The lump disappeared, and he rapidly recovered.

Such an accumulation of fæces in the cæcum often occurs when the patient is getting well. I remember one of the officials of this hospital had perityphlitis some years ago, and had made a good recovery. He had been about for some days, and then Dr. Stephen Mackenzie, who was then resident medical officer, asked me to see him again, for the man had still a lump in his groin. I examined it, and it was evidently due to fæcal accumulation, for it disappeared as the patient got stronger.

It seems that the inflammation may leave a catarrh of the mucous membrane of the cæcum, and perhaps also of the ascending colon. In some cases this gives rise to diarrhœa, and in others the inflammation weakens the muscle, and fæcal accumulation is the result.

LECTURE XLI.

PERITONITIS.

WHAT are the morbid appearances in death by peritonitis? The appearances are typical of those of inflamed serous membranes in general, so I will ask you to pay particular attention to this point. In cases in which death has occurred at a very early period of the inflammation, as, for instance, in cases of death shortly after abdominal section, we notice two things, first, that the membrane has lost its natural bright and shiny appearance, and has become dull and lustreless; secondly, there is inflammatory congestion of the mucous membrane. The congestion of peritonitis is not uniform; we see red bands along the intestine, the bands having been formed at places where the round intestines were less closely in contact, and congestion was thus favoured.

If the patient lives sufficiently long after the onset of the inflammation, an exudation is in all cases formed. In former days it was customary to classify peritonitis as adhesive and suppurative, since in some cases a solid lymph, tending, if the case progresses favourably, to lead to adhesion, is formed, whilst in other cases we find fluid pus. The transition between lymph and pus is, however, gradual, and we commonly find firm lymph in some parts of the peritoneum, and pus in others, as it depends on the local intensity of the inflammation, whether the proportion of fibrin or of corpuscles in the exudation preponderates. As the exudation occurs the line of congestion on the gut disappears. If the patient lives for a few days we may find a very large quantity of pus in the abdominal cavity.

It is of importance to notice the colour of pus. A creamy yellow pus usually indicates that the peritonitis has arisen by extension of suppuration from some other suppurating area; if the pus is orange-yellow, it indicates that a perforation of the gall-bladder has been the excitant; a brownish-yellow colour implies that there has been perforation of the intestines and fæcal extravasation; if the pus has a muddy-greyish appearance there has probably been an admixture of chyme, and you should search carefully for a perforation of the stomach or duodenum. I lay stress on these particulars, for it is sometimes difficult, except from such considerations, to determine what has excited the peritonitis; the bowel is exceedingly soft, and in turning it over we may easily tear it, and mistake such tears for natural perforations. Attention to the colour of the pus will tell us in what direction to look, and may tell us what has set up the peritonitis, even if we cannot find the actual place where it began.

What does this softening of the bowel mean? It means that the inflammation has extended from the peritoneum into the substance of the bowel. One great danger of peritonitis is, that the inflammation tends to extend in this manner, and to paralyse the muscle by cutting off the nutrient flow. Acute inflammation softens all tissues. But, you will tell me, an inflamed lung feels hard. That is true, it feels hard, because the tissue is tightly packed with exudation, but if you thrust your finger through it you will feel how soft it is. It is not hard, it is only tight.

The wall of the intestine is then softened, and the peritoneum is partially separated from the muscular coat. How do we know this? Because we can easily strip the peritoneum from the muscle which it covers. The meshes of the tissue are packed with exudation, and this exudation compresses the blood-vessels, and stops the flow of lymph in the tissue, so that the muscle becomes paralysed and is unable to resist the pressure of the accumulating fluids and gases. Thus the bowel becomes more excessively distended. Under these circumstances it is very probable that a portion of the wall may soften down and become perforated; perforation is one of the great dangers of suppurative peritonitis, for the inflammation is extremely violent, and, moreover, no conservative adhesions can take place. Further, the pus is extremely irritant, and its very presence tends to keep up the inflammation. Having learned these facts from the study of morbid anatomy, I used many years ago to ask the surgeons why they did not wash out the peritoneum, and they used to shrug their shoulders, and say it was impossible. Yet, as you know, this operation now saves many lives.

A special form of peritonitis is known as gangrenous peritonitis. There is much purulent lymph, but it may be only in a limited area of the abdominal cavity, and we notice that a very limited portion of the intestine lying amongst the exudation has a dark, reddish-purple appearance, and you see that a limited area of this portion is greenish-brown, the colour of sloughing tissue. On taking hold of this purple piece of intestine you are impressed by its leathery feel, and it is evident that it is thickened by very tightly packed exudation. There has been some obstruction to the circulation, but why has it been so limited? On examining closely you find that the purple part does not shade into the surrounding grey intestine, but the two are abruptly separated, and you know at once that there has been hernia, or obstruction under a band; there is no doubt that the piece of bowel has been strangulated.

In other cases we find three or four portions of gangrenous bowel in the ileum, near the ileo-cæcal valve. You open the intestine and find typhoid ulceration of the bowel.

Perhaps, when you open the abdomen you find this condition. There is purulent lymph and pus widely diffused in the abdominal cavity; there is extreme congestion, the whole peritoneum is fiery red; the bowel is very soft, so that it tears at a touch, and in places may be gangrenous. When you meet with these appearances you may be confident there has been either violence or perforation. It may be that there has been a kick in the abdomen, or a violent wrench, or it may be there has been perforation, and you must not overlook that the perforation may have been due to swallowing an irritant poison.

I. Peritonitis is never idiopathic, or so rarely idiopathic that we are justified in saying never. I mean by this that there is always a traceable antecedent. Peritonitis is never merely the result of cold or of slight injury. Cold may determine it when there is a tendency to phthisis, but not in a healthy person. When you find that peritonitis has arisen apparently from some slight cause, look for some latent condition which has really given rise to it. It may be cancer, it may be phthisis, or, in an infant, congenital syphilis. Remember that a child may be born with syphilitic peritonitis.

II. Peritonitis most commonly arises from extension of inflammation from some other organ.

a. From the uterus, especially in the septic conditions common after delivery.

b. Gonorrhœal inflammation may extend in the male to the bladder, and thence to the peritoneum ; in the female it may take this course, but more commonly spreads by way of the uterus.

c. Decomposing urine may set up cystitis, and from this by extension peritonitis may arise.

d. Injuries to, or operations involving, the perineum.

e. Disease of the bones of the pelvis. In fact, peritonitis may arise in all forms of pelvic inflammation, and this is a danger we must never overlook.

f. By extension of enteritis, most commonly in connection with typhoid fever.

g. It may arise by extension of inflammation from the kidney, but this is rare. In fact, there is no organ in the abdomen disease of which may not give rise to peritonitis.

h. Inflammation may extend from the pleura to the peritoneum, but this is very rare indeed.

III. I have told you that peritonitis may arise from direct violence, and there is a special likelihood of its occurrence in cases of penetrating wounds of the abdomen, whether accidental or made by the hands of the surgeon.

IV. Peritonitis may occur in connection with rheumatic fever, but this is exceedingly rare.

V. The most severe and rapidly fatal form of peritonitis arises when poisonous ingredients are poured out into the peritoneum. The following are the most common ways in which this occurs :—

a. Perforation of the stomach by round ulcer.

b. Perforation of the stomach by corrosive poison.

c. Perforation of a typhoid ulcer of the intestine.

d. Perforation of cæcal appendix.

e. Rupture of an abscess into the abdominal cavity. Most common are, liver abscess, arising either from tropical hepatitis or from a suppurating hydatid; and pelvic abscess in women after childbirth.

VI. Peritonitis, with or without other morbid changes, is found in many of the cases classed as puerperal fever or puerperal septicæmia.

VII. Peritonitis arises in connection with morbid growths of the peritoneum; these are tubercle, cancer, hydatid, and syphiloma.

VIII. Certain poisonous conditions of the system may give rise to peritonitis. It is common in Bright's disease, and is then usually suppurative, and may also occur in secondary syphilis.

IX. Protracted venous congestion may give rise to peritonitis, as it may to pleurisy. Hence peritonitis may come on in failing heart, or in ascites due to cirrhosis of the liver.

Let us now turn to consider the suffering in peritonitis. I told you that in some cases of pleurisy the onset is obscure, and the same is true of peritonitis. In cases of tuberculous and cancerous peritonitis the onset is commonly insidious, and the pain not severe.

Much more commonly peritonitis begins with severe pain, and the pain itself tends to kill the patient rapidly. This is especially the case where part of the alimentary canal or the gall-bladder have been perforated. The breathing quickly becomes embarrassed, and this for two reasons: first, that the downward movement of the diaphragm moves the inflamed peritoneal surfaces, and thus increases the pain; and second, because pain, irrespective of its seat, tends to hinder the breathing.

The pulse in peritonitis is characteristic. When I was a student preparing for examination we used to ask each other questions, and a favourite one was, "What is the pulse of peritonitis?" the answer being, "Small, hard, and wiry." It is small because the heart throws only a small quantity of blood into the arteries at each systole, and it is hard and wiry because the irritation causes the peripheral arteries to contract, and so raise the blood pressure.

The patient lies usually on his back, with his legs drawn up, in order to lessen the tension in his abdominal cavity. He vomits obstinately everything he takes into his stomach, and in consequence of this soon comes to suffer terribly from thirst. As the inflammation usually involves the intestinal muscles, there is complete constipation. The paralysed bowel gives way to the pressure of the gases which accumulate in the intestine owing to the passive obstruction, and the belly becomes extremely distended—tympanites. Persistent hiccough is common. The face becomes pale and bluish, the cheeks are pinched, and the eyes are sunken, these making together the appearance so suggestive of peritonitis or of some other acute disturbance in the abdomen. The pallor arises because there is little blood in the arteries, the blueness because that blood is insufficiently aërated, and the features are sunken because, owing to unsatisfied thirst, the water in the cellular tissues has been withdrawn into the general circulation. The body is commonly bathed in a cold sweat, and the breathing is shallow and heavy. Gradually the patient becomes pulseless and dies.

I have told you the morbid appearances of the inflamed peritoneum, and must now add a word or two as to the other organs. We find that the left side of the heart is empty and contracted, while the right side is extremely distended. The lungs commonly weigh less than normal, containing plenty of air, but little blood. If we ask, What was the immediate cause of death? the only answer can be, Pain and irritation. Remember that there is a degree of irritation that is worse than pain. The patient suffers more than he can bear, and it yet is not from pain. He throws himself about and groans—in a word, is terribly restless.

In peritonitis the chief things we are called upon to treat are symptoms, the cause being beyond our control, as a rule, by the time peritonitis has come on. We have, as far as possible, to subdue pain and restlessness, and this may be done to some degree by warmth to the abdomen, by opium, and by alcohol. We have to try to relieve thirst, and we give the patient ice to suck, and if he is quite unable to retain fluid given by the mouth, we may inject cold water into his rectum—a few ounces only at a time. The distension sometimes is the chief trouble, and this may sometimes be relieved by passing a long tube up the rectum; if this does not do good, we may tap the intestine through the abdominal wall with a fine trocar.

Remember that in cases in which there is much pus in the abdominal cavity the chemical irritants contained in the pus are absorbed, and appear to kill by paralysing the heart, and in some such cases you may save life by washing out the peritoneum.

LECTURE XLII.

JAUNDICE.

Let us call to mind that the liver has two leading functions, one is a clearing away of the past, a cleansing of the body, and the other is a working up of new material. If it should happen that the first of these, the excretory function of the liver, is hindered, we should expect that the body would become choked with dead material and become dulled, gloomy, and tending to stagnate. If, on the other hand, the second of these two great functions, the secretory working of the liver should be hindered and tending more and more to fail, we should notice that the freshening process, leading to the new formation of blood, was languishing—we should see a losing of the healthy redness of blood, a losing of the eager healthy appetite, nutrition failing, flesh and muscular power being lost. If, when thinking of liver disease, you will bear those facts in mind, it will save you much trouble, and it will be easy for you to grasp the symptoms of such disease.

One of the commonest morbid conditions of the liver is venous congestion, and it is very instructive to watch the symptoms of such congestion, for they reveal how essential it is for the healthy action of the liver that the blood should flow freely through it.

Venous congestion of the liver most commonly arises from heart disease, but it may be secondary to vesicular emphysema, or to some other chronic condition hindering the flow of blood through the lungs. To some extent it occurs also in brain failure, but that is comparatively little marked. It is present in some degree whenever breathing fails, as in fevers.

What are the symptoms of venous congestion of the liver?

The first noticeable condition is evidence of increased tension—a sense of swelling about the liver; this swelling and tension culminating in more or less pain and tenderness. As this takes place, indigestion becomes a marked symptom, and some degree of jaundice frequently supervenes. The skin becomes more or less yellow, and the urine contains bile, and often lithates.

What are the changes in the liver as venous congestion becomes more and more extreme? The capillaries of the liver become extremely distended with blood, and enormously dilated, and the liver cells become infiltrated with an albuminous granular material. This granular change is a condition very commonly found after death by pneumonia, by burns, by fever, and other acute disease. Moreover, where there is extreme venous congestion, hæmorrhage takes place around the capillaries among the liver cells, still more breaking up these, thus producing the purple hæmorrhagic areas so commonly seen in nutmeg livers; the liver cells, where not broken up by hæmorrhage, are much compressed by the dilated capillaries, until it is difficult to define them, and they become charged with bile-pigment. You will see all these changes in the nutmeg liver.

If you are asked the cause of jaundice in nutmeg liver, your answer should be, "Compression." The liver ducts are compressed owing to the swelling of the tissue, and the bile cannot pass freely. The mucous membrane of the common bile-duct is also swollen, and its orifice into the duodenum is compressed by similar swelling, and thus arises a further hindrance to the passage of bile. What must be the result if the bile cannot escape? The body must become charged with a poison, and this will be an increasing source of weakness. It has been noticed that, as the liver swells, the weakness increases.

What do we mean by jaundice? If there is a yellow look in the skin, and a yellow tinge of the conjunctiva, do we always speak of such a case as jaundice? No, when we speak of jaundice, we usually mean a condition to be referred to disease of the liver, whilst such a yellowness is not uncommon in cases of malarial poisoning. Again, many a young woman is taken to the doctor under the impression that she is suffering from liver-disease, and the cause of her yellow complexion is disintegration of the blood—anæmia. The disintegration of the blood that occurs in pyæmia is often attended with yellowness of the skin and conjunctivæ, and high-coloured urine, and in these cases it becomes difficult to know whether we should consider that there is jaundice.

We usually agree to regard as cases of jaundice, only those cases of yellowness of skin and conjunctivæ in which bile can be detected in the urine. What is the significance of the presence of bile in the urine? When death occurs in such cases we find all the organs and tissues stained with bile. The blood is stained like all the other tissues; we find the lining membrane of the blood-vessels, and the clots in the heart, stained with bile. The least visible staining is that of the nerve structures—we are often curiously impressed by noticing how little the white matter of the brain is stained. If, then, the skin is bile-stained and we find bile in the urine, we know that all the tissues are charged with bile, which they have taken up from the circulating medium, the blood. The next question that arises is how the blood became contaminated with bile, and this is a question which has very much puzzled pathologists and physiologists, and which opens up the question of the natural origin of bile.

In very many cases jaundice undoubtedly arises from obstruction to the outflow of bile after its formation by the liver, but in other cases of jaundice which have terminated fatally, we find on post-mortem examination that the large ducts are entirely free from obstruction, nor is there any condition of venous conjestion which could have given rise to jaundice in the manner I described just now. There are several conditions which we know from clinical and pathological experience can give rise to the presence of bile in the urine, and to bile staining of the skin; and in fatal cases we find no evidence of obstruction to the flow of bile from the liver. These conditions are—(1) Poisoning with phosphorus, and in certain cases with alcohol and with chloroform. (2) After the bite of certain snakes. (3) Certain febrile conditions associated with poisoned blood, as pneumonia, typhus and typhoid fevers, especially the former, pyæmia, relapsing, malarial, and yellow fevers. (4) Acute yellow atrophy of the liver. (5) In some very exceptional cases, shock, or fright, have caused fatal jaundice. Now, in all these cases, it appears that there must have been some disintegration of red corpuscles—a death in blood—from which the jaundice has arisen. It is not possible for me fully to discuss this subject here, but I shall describe acute atrophy in a later lecture. We now pass on to consider the commoner cases of jaundice in which there is obstruction to the outflow of bile, so that it is absorbed into the blood after its secretion by the liver.

I have already spoken of one variety of this kind of jaundice—namely, the jaundice, usually slight in character, which results from venous congestion of the liver, and gives rise to the yellow tinge of the skin so commonly met with in mitral valvular disease, and other conditions of heart failure. There remain two broad classes of cases to consider, those in which the obstruction to the flow of bile arises from catarrh of the bile-ducts, and those in which the obstruction is mechanical, absolute, and in many cases permanent and irremediable. Cases of the former kind are usually known as catarrhal, or simple jaundice, those of the latter are called mechanical jaundice. We will consider first catarrhal jaundice.

In catarrhal jaundice the obstruction to the flow of bile arises because the mucous membrane of the ducts is swelled, and the narrowed passages are occluded by condensed mucus. The depth of the yellow colouring is never so great as in the mechanical variety. Simple jaundice arising from uncomplicated catarrh of the ducts never kills; if therefore death has occurred in a case of jaundice, and on post-mortem examination you find the jaundice was due simply to catarrh of the ducts, look for evidence of some gross organic disease to which the catarrh of the duct is secondary. In the cases in which recovery occurs, the catarrh of the bile-ducts usually arises by extension from the stomach and duodenum. In such cases the primary catarrh of the stomach and duodenum may have arisen from very various causes, such as a weak, exhausted state of health, overwork, fatigue, or exposure to a foul atmosphere. The gastro-duodenal catarrh set up by abuse of alcohol is another cause. When death occurs from catarrhal jaundice we look for evidence of organic disease. The catarrh may have been due to prolonged venous congestion of the mucous membrane of the ducts, such congestion as comes on in chronic heart and lung disease, but jaundice is not common in phthisis. The catarrh may have arisen by extension from dysentery or other severe form of enteritis. We may find that cancer, or non-malignant ulcer of the stomach, have led to the catarrh, or we may find cirrhosis of the liver. Where jaundice—I do not mean merely a cachectic, pale yellow colour of the skin—occurs with cirrhosis of the liver, it is secondary to catarrh of the stomach. Usually, also, when jaundice occurs in connection with hydatid, it is due to extension of catarrhal inflammation to the duct, but hydatid may also cause jaundice by direct pressure, or we may find a number of the smaller ducts blocked by hydatids.

Mechanical jaundice may then be due to hydatid, but this is one of the rarer causes. When in jaundice the tint of the skin is very deep—a greenish-yellow, or, it may be, almost bronze—we look for a cause of long-continued obstruction. The deeper the tint, the more strongly does it point to complete and permanent obstruction. We see the deepest tint in cases of cancer of the duct, medullary carcinoma, beginning along the lesser curvature of the stomach underneath the liver, or in the head of the pancreas (this is comparatively common), or in some other part adjoining the common duct. We find the duct filled up by cancerous growth, and completely blocked; it may be merely pressed upon from outside, but it is more commonly filled by growth. The obstruction may be due, not to cancer, but to the pressure of a great mass of strumous glands lying under the liver. The obstruction may be due to gall-stones. Worms have been mentioned as a cause, but I have myself never met with such a case. If, then, you are asked, "What are the chief causes of mechanical jaundice?" answer, "The commonest is cancer. Gall-stones are a frequent cause. It may be due to the pressure of enlarged glands on the duct." In later lectures I shall describe the distinctive features of cancer of the liver and of gall-stones, and shall speak of cirrhosis and of acute atrophy of the liver, and all that remains for our consideration in this lecture is the effect of mechanical occlusion of the duct, and of long-continued jaundice on the liver and on the rest of the body respectively.

When the common bile-duct is completely blocked, bile accumulates in the ducts behind the obstruction. How do we know this? We find it on post-mortem examination. In moderate cases the common duct is much dilated by the accumulation, whilst in extreme cases the small ducts are also much dilated, until the liver is riddled like a honeycomb with dilatations of the ducts filled with condensed bile. The liver is considerably swelled, but is usually smooth on the surface. Under the microscope we see that the liver cells are charged with bile, and that they have been much destroyed by pressure. Further, in examining the bodies of such patients as have died with severe jaundice we commonly find evidence of recent acute inflammation in various parts of the body. Accumulated bile in the tissues acts as an irritant, and may excite bronchitis, pneumonia, nephritis, pleurisy pericarditis, peritonitis—in a word, inflammation in any part of the body. We see this strikingly marked in the skin; first there is itching, and then eczematous eruptions appear, the skin becomes sore by scratching, and a tortured, inflamed surface results.

If, then, you are asked, "For what complications would you be on the watch in a case of jaundice?" answer immediately, "Inflammation." Take the temperature day by day, but bear in mind that in inflammation of a mucous membrane the temperature is but little raised, so that bronchitis or nephritis may come on without any warning rise of temperature. Apart from the liability to acute inflammation, what are the common symptoms? There is increasing weakness. When the blood is charged day by day with an excretory effete product, the dead material in it tends to kill the blood more and more, then muscle loses its contractile power, nerve loses its nervous power, mind gets darker and gloomier, until the patient dies. We see such symptoms alike in cases of retained urine and retained bile.

Finally, in cases in which the duct has been blocked for months, and the liver-substance has been much destroyed, as already described, by the distension of the smaller ducts with condensed yellow bile, there is a liability towards the end to the onset of symptoms like those of acute atrophy—that is to say, an exceeding asthenia, with delirium and death. There is always a risk of this when the duct is completly obstructed. It may therefore occur in cancer, but is not common; we see it more frequently in cases of gall-stone. I have not known it to occur in young persons. It is especially liable to come on where there has been much abuse of alcohol, for the tissues of such persons have a tendency to rapid decay; where the nervous and muscular organization have been much weakened by any cause there is also a special likelihood of its occurrence. The history of such a case is commonly this: in an elderly person there has been repeated jaundice due to obstruction by gall-stones in the common bile-duct, and at last the jaundice becomes persistent, extending over months, and the liver becomes enlarged. The patient complains of increasing—somewhat rapidly increasing—weakness, followed by delirium and typhoid symptoms, ending in death.

LECTURE XLIII.

GALLSTONES.

If a middle-aged or elderly person is seized with pain in the right hypochondrium, quickly becoming severe, and radiating more or less to other parts, accompanied by vomiting, usually constipation, and a greater or less degree of collapse, the attack lasting for many hours, we suspect that a gall-stone formed in the gall-bladder has passed into the excretory duct, and is giving rise to biliary colic. If such an attack is followed by jaundice, our suspicions are confirmed. It is, however, important to remember that symptoms closely resembling those above described may arise, though far less frequently, from another condition—namely, morbid growth. As a result of many years of experience, I am in the habit of teaching that any morbid mass situated near the common duct may, from time to time, by inflammatory or other change, swell, and so press upon the duct, and lead to symptoms somewhat resembling those of gall-stone.

Gall-stone trouble usually takes one of two forms. An accumulation of gall-stones in the gall-bladder may set up irritation passing into inflammation and suppuration. The gall-bladder may be perforated, and pus poured out into the peritoneal cavity, exciting fatal peritonitis, but more commonly adhesive inflammation occurs, and the pus makes its way through the skin, or into the stomach or intestine. The symptoms in these cases are generally pain and tenderness, more or less persistent, in the region of the gall-bladder, and after a while we feel a marked swelling in this region.

The other form is the more common, where a small gall-stone passes down the cystic duct into the common duct. It may pass into the duodenum, we are led by the symptoms to think in a few hours, but it may take days, or months. I had a man under my care in the hospital who had had jaundice for four years. I was doubtful about his assertion, but his family medical attendant confirmed his statement. Eventually symptoms very like those of ague came on—violent rigors recurring again and again for many months. At the post-mortem examination I found a gall-stone, which had been formed in the gall-bladder, had set up ulcerative inflammation in its wall, and so made its way through into the common duct. There was a large gall-stone lying half in the gall-bladder and half in the common duct, and the protracted irritation had set up suppurative inflammation of the common duct, which had travelled along the hepatic duct into the liver-substance, and riddled it with abscesses. The recurring suppuration was the cause of the shivering. Such a case is extremely exceptional. In some cases gall-stones may set up fibrinous inflammation ; in others, chronic inflammation in the capsule of Glisson, leading to cirrhosis, may ensue, but such cases are also rare.

In cases of impacted gall-stone do not give a discouraging opinion. I will tell you why. It is astonishing for how many months the jaundice may persist, and yet the patient entirely recover. I used to say the patient might recover after the jaundice had lasted three months, and later I said six months, recently I had a woman under my care in the hospital in whom the jaundice completely diappeared after persisting for nine months. In such cases there is usually evidence that the duct is not completely blocked. We notice that the motions are not of the pipe-clay colour seen where the bile is entirely cut off from the intestine, but have a yellow tinge of varying intensity. Moreover, the colour of the skin is not the greenish-yellow, almost bronzed, tint seen when the duct is absolutely blocked, but it is a brighter yellow. If the duct is not completely blocked there is, of course, less likelihood that serious structural changes in the liver will ensue, since the pressure in the bile-ducts will not become so great, nor are the tissues so intensely poisoned with bile, nor does the system suffer so much from the malnutrition consequent on the absence of bile from the intestine.

There is another striking feature in cases of impacted gall-stone, the calculus may remain impacted, and yet the symptoms may rapidly disappear. I have known jaundice to disappear, and the pain to be absent for weeks, and then the jaundice recur when the patient had another attack of hepatic colic; these attacks kept recurring for years. In one case the jaundice disappeared, but the patient had an attack of hepatic colic almost weekly. The general history of the case, course of symptoms, and subsequent recovery, indicated that the stone was still in the duct, but that the bile could get past it; hence the jaundice disappeared, but the recurrence of paroxysms of severe pain manifested that the stone was still lodged. It is important to recognise that the jaundice may disappear, although the stone has not got completely out of the duct, for in such a case the stone may be ulcerating its way through the wall of the duct, and may open a way into the peritoneal cavity; if bile flows into the peritoneal cavity, a virulent form of peritonitis results. We see then that, even when the jaundice has disappeared, we must be cautious to keep the patient at rest for a time. If the patient be kept at rest, the stone usually passes safely, whilst if he be allowed to get about too early, conservative adhesions may be torn away, and death rapidly result from peritonitis.

In treating a case of prolonged jaundice from the supposed impaction of a gall-stone, the first thing we have to do is, then, to keep the patient at rest, so that if the stone is ulcerating through the wall of the duct, adhesions may form, and peritonitis be averted. What is the next point to consider? Keep the patient's blood as fresh as possible, or he will lose his appetite, lose his desire to live, and get weaker and weaker. How are we to keep the blood fresh? Do not take too limited views with regard to food. What is food? The products of the earth to renew the body; the products of the animal and vegetable worlds are taken by us into our bodies in order that we may renew our blood. One of the most marked errors I have seen in the treatment of liver cases has been too much keeping to milk, fish, and things of that kind. Let us vary the patient's food as much as we can, that we may freshen his blood. If we get new materials into the blood breathing and respiration are promoted, and thereby oxidation is promoted. Now in cases of this kind it is most important to promote oxidation, for experience has shown that gall-stones are especially found in persons who have been leading too sedentary lives. Gall-stones are mostly met with in women at or after the middle period of life, who, owing to the cares of house, have led very monotonous indoor life, not sufficiently under the influences that oxidise and strengthen, and whose excretory powers have become enfeebled. I have known gall-stone colic occur in a young man, but this is exceedingly rare. If then we want to prevent the formation of gall-stones, and to remove those already formed, we should go for oxidation. Have we any means of promoting oxidation by giving drugs, so that we can in this way assist persons whom it is necessary to keep in bed for a time? Physiologists tell us that alkalinity of the tissues promotes oxidation, and we therefore give a little alkali—some bicarbonate of potash or ammonia. Another great means of promoting oxidation is to get away as much effete material as possible, for the presence of waste products in the tissues checks the activity of their chemical processes. What are we to do in such cases as these, in which, the bile-duct being more or less completely blocked, we cannot expect to get the bile very freely down into the intestine? We have skin, we have kidneys, we have lungs to work upon, all these are excretory organs. Give plenty of water to drink, so that the poison may be washed out of the body, and the organs not become so choked with bile that acute inflammations come on. Aid the action of the water by keeping the air of the sick-room sweet and fresh. Can we usefully act on the intestine? I have told you that in many of these cases some amount of bile gets past the stone, so that by giving aperients we should promote the rapid discharge of this small quantity, but can we do anything more? We can have little doubt that there is a physiological co-ordination between the action of the duodenum, and the action of the gall-bladder and the bile-ducts. This is what we should expect from theoretical considerations, and practically we know that when the intestine takes on increased peristaltic action, a greatly increased quantity of bile comes down. We may then reasonably infer that by the use of gentle aperients we can do something towards drawing the stone out of the duct into the duodenum. We therefore give a little calomel occasionally, or we may give a little carbonate or sulphate of magnesia, or better, a warmer aperient, a little rhubarb or senna. We thus quietly feel our way day by day, remembering also that it is a relief to get the bowels a little moved, and that the patient likes it.

I will just add, be slow to discourage. With sensitive nervous patients, to keep up hope, and to guide them away from depression, is in itself a great help. Remember what I have told you, that a patient may entirely recover after the jaundice has persisted for many months. Even when patients have had attacks year after year, it is astonishing how they get rid of them. Promote rest, so that the stones in the gall-bladder may get set, impacted in the gall-bladder, so that they cannot be shaken and dislodged into the common duct. I have, by post-mortem examinations, been led to think that this is the way in which patients become freed from repeated seizures. We have to remember that we commonly find gall-stones in the gall-bladder, and yet there is no history to indicate that they have caused much trouble; therefore post-mortem examinations have taught me that rest should be the aim in treatment.

LECTURE XLIV.

ACUTE ATROPHY OF THE LIVER.

In acute atrophy of the liver we have a remarkable demonstration of the mysterious interaction of the liver with the nervous system. It has long been recognised that fright may cause jaundice, and that mental shock or worry may lead to great disturbance of the liver. People who are getting mentally depressed go to medical men and inquire about their livers. Patients suffering from all kinds of things, perhaps going wrong in their businesses, perhaps going wrong in their heads, will come to the doctor and say, "Doctor, do you think it is my liver?" I hear that again and again, almost every day. A young medical man came to me. His father had been a hard drinker. This man did not drink, but he was afraid of the sins of his father, and afraid that he might take to drink. "Do you think it is my liver?" he said to me.

In acute atrophy of the liver, the liver rapidly shrinks. Instead of weighing two-and-a-half to three pounds, it may shrink to about ten to fifteen ounces, and it becomes much thinner. The way in which I usually demonstrate this is by taking up the liver, and showing how it bends and folds over the hand. It has a drab-red colour. When it is cut into, the most striking feature is the loss of lobular appearance, though here and there a little remains. It has become homogeneous-looking. It is of about the normal consistency. In colour it reminds us of a piece of turkey rhubarb—deep red and pale yellow blending. The ducts are completely open. On examination by the microscope it is a very impressive sight—I may say, a bewildering sight. The liver-cells have disappeared, and are replaced by a homogeneous and finely granular material, and this alteration is widespread—in fact, throughout the liver. We see the capillaries distinctly, and the liver-tubes, but the cells have disappeared; it has been spoken of as a " solution of the liver," as if the liver-cells had dissolved down into a granular homogeneous substance. We can find no evidence of inflammation, of hepatitis; in this respect it is a most perplexing disease. It is an acute death in the liver, and atrophy is the best name to give to it.

On post-mortem examination we find that the cases are to be arranged in two groups—primary and secondary. The secondary cases are those in which we find chronic change in the liver, it may be cirrhosis of the liver, or the dilatation of the bile-ducts and destruction of much liver-tissue by pressure, which occurs when there is long-continued complete obstruction of the common bile-duct. In the primary cases there is no appreciable antecedent organic disease. There may have been an antecedent condition of great exhaustion, such as recent confinement, great exhaustion of nervous energy, or abuse of alcohol; but in numerous cases no such antecedent condition is discoverable. When we examine the body in such a case the jaundice may be remarkably slight—sometimes it requires careful examination to detect it—but the body is usually wasted, the wasting being rapid and recent. All the organs, with the exception of the liver, seem free from structural decay, but they are bile-stained. The body is usually that of a young person, from fifteen to thirty years of age. When we examine the liver, we find the appearances I have already described. In the secondary cases there is not the same striking immunity from organic changes, and the morbid appearances of atrophy are complicated with the changes I have already described in lecturing on jaundice, or those of cirrhosis, of which I shall speak in my next lecture.

Clinically, we find that the primary cases can be arranged in two groups. There are some cases in which the first warning of the disease is great distress in the nervous system. There is restlessness and delirium. At first it is doubtful if there is jaundice, but in a few days it is beyond question. A young girl was found by her mother standing in a corner of the room naked. She had become delirious during the night. In two or three days there was slight but distinct jaundice, and soon typhoid symptoms came on, and she died of acute atrophy.

As an example of the second mode of onset of acute atrophy of the liver I may give the case of a young, well-nourished woman who came to the out-patient department. The case appeared to be one of simple catarrhal jaundice, but she vomited a good deal, and I could not understand why, as there did not seem to be any affection of the stomach. On account of the vomiting I thought it wiser to admit her to the hospital. In few days there were symptoms of acute atrophy, but she had then been suffering from jaundice for several weeks. We see, then, that some cases follow what appears to be simple catarrhal jaundice, while in other cases jaundice appears after the onset of delirium.

These patients rapidly become extremely asthenic, pass into a typhoid condition, with muttering delirium, and die usually in the course of a week or two. The temperature is variable ; it may be subnormal or it may be raised, the latter especially towards the last. The great guide is the condition of the liver dulness, which gets smaller and smaller, and as it shrinks there is increasing stupor, passing into coma. During the progress of the disease we get evidence that the processes of oxidation in the body are diminished. In the urine the urea is diminished, and its place taken by leucin and tyrosin. The extreme loss of muscular and nervous power also tell us that oxidation is greatly diminished.

Before closing this lecture I may say a few words about fatty liver. This is very common as a large soft, heavy liver, with a thick rounded edge, but of much the same colour as the normal liver. We meet with this as a result of excessive beer-drinking. When fatty liver is spoken of, a pale-yellow liver is usually meant—otherwise the characters are much the same as those already given—the liver has a smooth surface, a thick rounded edge, and is heavy. When we cut into it, it is very soft, like suet. Under the microscope we see that the liver-cells are charged with fat-granules.

Where I have met with fatty liver it has been in cases from the surgical wards. Why is this? They have been kept in bed for months, given a large amount of food, and a certain quantity of alcohol, and in that way the liver has been choked up.

That is how they make the livers for the pies in Strasburg—they tie the geese up and stuff them with food.

There may be a complexity of diseases in the same liver—gummous masses from syphilis, cirrhosis from excessive spirit-drinking, fatty change from excessive beer-drinking, and lardaceous disease, all at once.

LECTURE XLV.

CIRRHOSIS OF THE LIVER.

Cirrhosis of the liver is connected with, though not, in my opinion, entirely due to, abuse of alcohol. Moreover, there are several other forms of contraction of the liver besides alcoholic cirrhosis, so-called. It will therefore lead to greater clearness if, before describing to you the last-mentioned disease, I refer briefly to the other forms of contraction of the liver. This will enable you at the bedside to avoid speaking of cirrhosis of the liver, when there is really only evidence of contraction. To avoid the possibility of this error, Murchison did not speak of cirrhosis, and used only the clinical term, contraction of the liver.

It has for many years been recognised that if venous congestion continues long, the connective tissue of the liver swells, and undergoes a process of stiffening and thickening. If, therefore, you are asked, "Does heart-disease cause an early degree of cirrhosis?" you should say, "Yes." But if you are asked, "Does it cause an extreme condition of cirrhosis—the hob-nailed liver?" say, "No." At least, I have never known such a condition come on in heart-disease, except where there has been abuse of alcohol also. But do we not, in some cases of chronic heart- and lung-disease, such as lead to extreme and long-continued venous congestion of the liver, and in which there is no evidence of the abuse of alcohol, meet with such dropsy as is usually found with hob-nailed livers, with a great degree of ascites, and very little œdema of the legs, indicating that the venous circulation within the abdomen has failed to a much greater degree than that in the lower extremities? Such cases are by no means rare. What is the cause? Contraction of the liver. In chronic venous congestion of the liver there is a liability to the onset of inflammatory changes in the capsule of the liver—perihepatitis. The capsule becomes thicker, and then the thickened material contracts and squeezes the liver extremely, until it may be only a third of its natural size, and the portal circulation is much obstructed.

There is another class of cases in which thickening of the capsule is combined with thickening of the connective tissue of the liver, so that the left lobe may be destroyed and the liver transformed into a square mass. These cases are not necessarily alcoholic, but may occur under conditions which we cannot explain, and in many cases the disease affects quite young persons.

There is a form of contraction due to simple chronic atrophy of the liver, in which the liver-substance wastes away; contraction due to extreme fibroid change in the capsule and substance of the liver, without much unevenness of the surface—a uniform fibroid change. Fibroid change may be due to a long-impacted gall-stone, leading to inflammation and thickening of the connective tissue of the liver. There is a class of cases in which there has been tubercular change in the peritoneum, extending over many months, accompanied by tubercular formation in the liver, and leading to great fibroid thickening of the connective tissue, and contraction of the liver, like cirrhosis. Tertiary syphilitic changes also produce fibroid contraction, and in this condition difficulty in diagnosis is most likely to arise. The question whether a given case of hepatic disease is one of cirrhosis, tertiary syphilitic fibroid change, or cancer, is one to which sometimes no positive answer can be given until the progress of the case has been watched.

What are the common effects of all varieties of contraction of the liver? (1) Failure of nutrition, and loss of muscular and nervous power. (2) Great liability to catarrhal inflammations of mucous membranes. (3) Increasing change in the urine. (4) After a time signs of obstruction to the portal circulation supervene. The belly becomes greatly distended, but the percussion-note is resonant. This gaseous distension is the prelude to the onset of ascites.

Since the typical hob-nailed liver is most commonly met with in persons who have been great spirit-drinkers, it becomes important, for the sake of clearness, to differentiate the symptoms properly due to the liver-disease from those which are the direct result of the abuse of alcohol.

When there has been excessive drinking, one of the marked symptoms is weakness in muscular and nervous function. Failure in the nutrition of mucous membrane is common, as we may see in the throat, the tongue, the lips, the rectum, and even the urethra. In any one of these situations catarrhal inflammation may come on. Extreme catarrh of the throat, followed by superficial ulceration, always leads us to think of alcohol. If a patient has gleet of the urethra, and there is no evidence of gonorrhœa or of tubercular disease, think of alcohol. If the patient complains of diarrhœa in the morning, think of alcohol.

When the liver is much congested by the abuse of alcohol, the appearance of the urine is very characteristic. It affords evidence that the red corpuscles of the blood are broken up in excessive numbers, the urine is very high coloured, and, owing to the presence of pigments, there is a purple coloration produced on the addition of nitric acid. The deposit of urates gives evidence of diminished oxidation. Soon catarrhal inflammation comes on; there is vomiting in the morning, mucus being brought up, and the patient is unable to eat in the morning, and there are other symptoms of chronic dyspepsia.

As months go on there is increasing evidence of failure of general nutrition; the circulation becomes weaker and weaker, and there is mental as well as muscular failure. In extreme cases there is evidence of cerebro-spinal disease, muscular tremor, spasm, and paralysis, ending in dementia. Epileptiform seizures are not uncommon, and in such cases we must be cautious not to give too gloomy a prognosis, since the cause of the disease is a removable one. In studying contraction of the liver we find such symptoms, due to abuse of alcohol, blended with those due to the fibroid change.

What are the effects of leaving off the alcohol? If tissue degeneration be not too far advanced, all these symptoms disappear. No more fits, no more tremor; the digestive power returns. The difference is astounding.

What do we see in acute fatal poisoning by alcohol? Paralysis of breathing. The breathing becomes more and more shallow, and as venous congestion of the brain increases, more and more difficult.

Before speaking of the suffering, we will describe the morbid anatomy of cirrhosis. We notice that the surface of the liver is no longer smooth, it is finely irregular, what is termed granular. On cutting into it we see the lobular structure with unusual distinctness, because there is a morbid formation between the lobules, isolating them. As we cut across the liver we feel that the substance grates a little, and we are sure that there is fibroid growth.

What do we find when the fibroid change is much farther advanced? There are circular projections on the surface of the liver, giving it the appearance known as "hob-nailed." When we cut into it we feel that it is much tougher than normal, and we see distinctly between the lobules a grey, semi-translucent, fibroid material. We notice that Glisson's capsule is thickened. The lobular substance is charged with fat and bile. The irregular surface, the toughness, and the appearance on section of the grey fibroid tissue, surrounding yellow areas of lobular substance, are most characteristic of cirrhosis.

In the most extreme form of cirrhosis the hob-nailed formation has much disappeared. The whole organ is contracted, and the surface appears finely granular, as in the early stage. The lobular structure has largely disappeared. The liver is extremely tough, and greatly reduced in size.

In another form of cirrhosis we find a similar toughness, a hob-nailed appearance, and much fibroid tissue, but a large liver. Instead of weighing a pound and a half, or thereabouts, the liver weighs perhaps four or five pounds. This condition has been called "hypertrophic cirrhosis," but the term is misleading, and further observation is needed before we can be in a position to speak positively about this condition. The only difference I have been able to find between these large livers and the contracted ones is, that in the larger livers there is a much greater fibroid growth. We find an enormous growth of spindle cells in the connective tissue of the liver, showing it to be a more rapid and malignant form of cirrhosis. On microscopic examination of the smaller livers, we see a fine felted fibroid substance, with here and there nuclei scattered. On post-mortem examination we always find cirrhosis of the liver associated with morbid changes in the stomach, with pigmentation, hæmorrhage, and thickening in the mucous membrane. It is not uncommon to find associated fibroid changes in the kidneys, the spleen, and sometimes in the lung.

We have now a better understanding than formerly of the pathology of cirrhosis, and can trace the history of the morbid changes in the liver. A new growth of spherical and spindle cells takes place in the connective tissue. Some have described it as occurring more particularly along the capillaries within the lobules—intra-lobular cirrhosis, but the growth much more commonly takes place along Glisson's capsule and around the circumference of the lobules—inter-lobular cirrhosis. I may here mention to you that in many parts we find that the fibroid changes begins mostly along the capillaries, whilst in others it is mostly along the adventitious coat of the arteries. We must not attach much importance to these differences. The spindle-cells become more and more closely packed, and by squeezing, more and more flattened, so that the original form of the cells and nuclei is much lost. In process of time there only remains a fine, felty-looking substance. It is this condition we mostly find after death from cirrhosis of the liver.

The fibroid material accumulates in very large quantities around the lobules, compressing the vessels, and so the power of circulation is increasingly cut off. As the fibroid material contracts, and the lobules are squeezed, the liver-cells atrophy and much disappear. In an extreme degree of cirrhosis we see as it were a sea of glistening fibroid material, with here and there an island of liver-cells. Owing to the accumulation, pressure, and destruction of vessels, the portal circulation becomes blocked, and blood accumulates more and more in the branches of the portal vein. The overcharged mesenteric veins relieve themselves by pouring out serum, and thus arises ascites. The overfilling of the splenic veins leads in some cases of cirrhosis to much swelling of the spleen, but in other cases the spleen is not enlarged, and in these it would seem that it has undergone a fibroid change, rendering it incapable of expanding. In the stomach we see also the results of extreme venous congestion—the connective tissue is thickened, the capillaries are much destroyed, and the mucous membrane is much pigmented.

Partly in connection with this condition of the mucous membrane of the stomach, partly owing to the deteriorated condition of the blood, arises one of the most surprising features in the suffering of cirrhosis. A patient may be going about much as usual, free from dropsy, when he begins to vomit blood —he turns suddenly faint and sick, bursts into a cold perspiration, and then brings up a large quantity of blood. In an hour he may bring up a like quantity, and this may be repeated several times. You may be called to such a case, and see in the room two or three basins into which the blood has been vomited. Some of the most severe and rapid hæmorrhage we meet with occurs in these cases of cirrhosis. It commonly comes on in cases in which there is no ascites, and I have known it kill the patient within twenty-four hours. In other cases the patient may pass a large quantity of blood from the bowel. These hæmorrhages are not necessarily fatal; I have known a patient with cirrhosis vomit blood again and again, and subsequently progress favourably.

What is the early suffering? The cases of contracted liver are always obscure for a while. Patients complain usually of symptoms of dyspepsia, failing digestion, fulness and discomfort after food, flatulence, and occasionally vomiting. With these symptoms we notice wasting. To account for these symptoms we must remember that the liver is one of the chief organs of nutrition, that its function is to convert food into blood, and that with the failure of this function will be associated deterioration of the blood, imperfect digestion, imperfect absorption, and wasting of the body. There will be cachexia—the skin loses its healthy red colour, and becomes more of a pale yellow. As the brain and nerve-tissues lack the supply of healthy blood there is an increasing feeling of weakness and of mental depression. The urine becomes high-coloured, and deposits the brick-dusty red lithates—such urine has long been recognised as pointing to liver disturbance. This shows us that there is failure of the great excretory function as well as of the great blood-forming function of the liver. When such symptoms persist month after month we infer that there is some serious degeneration of liver substance. Sometimes we have to ask, "Is it cancer of the liver?" Time answers that question. The morbid changes of cirrhosis progress more slowly than those of cancer.

Another fact impresses us. We notice that the stomach and intestines are becoming over-distended with gas, and from this we infer that the failure of circulation in the portal vessels is becoming so great that ascites will soon come on. As a rule after ascites has come on the blood tends to perish more rapidly, and there is liability to hæmorrhages in the skin, the peritoneal cavity and elsewhere. The next thing that we notice is, that the muscular system is losing its energy to an extreme degree. Death soon follows from heart failure or pneumonia, or it may be from nephritis or peritonitis.

In some cases there may be little or no ascites, but there is a markedly yellow colour of the skin. There may be little or no bile in the urine, the colour of the skin is therefore an evidence of death in the blood. The patient gets continually weaker and weaker, and more and more lifeless. There is weakness both of mind and body. These are the gloomiest cases of cirrhosis. The gums begin to bleed, there is bleeding from the nose, there is purpura, there is blood in the urine—the blood cannot stay in the vessels. Such patients rapidly die. We recognise from this that the great risk in cases of cirrhosis is that the blood will perish.

We come to the same conclusion if we look at the question from the other side. Patients are seen with extreme ascites owing to cirrhosis of the liver, we keep them at rest in bed, and the ascites disappears. I have seen this again and again, and it is a common experience with other physicians. How is it that with the liver much contracted the ascites can disappear? The accumulation of connective tissue blocks the circulation through the portal vein, hence the dropsy; but if it is simply the pressure of the fibroid tissue which causes the effusion, what leads to its disappearance? There is no doubt about the facts.

A man was at work, earning his living, when he met with an accident. He was severely injured, came into the hospital under the care of Mr. Jonathan Hutchinson, and rapidly died. I made the post-mortem examination, and found the most extremely cirrhotic liver I have ever seen; there was not a sign of dropsy, and the man had been able to earn his living, and yet he had the smallest and hardest liver I have ever seen. I remember standing beside the body and gazing at it with astonishment.

We are led to think that it is not the mere mechanical pressure of the fibroid tissue that causes the ascites. Further, if we can get rid of the ascites, the cirrhosis does not matter much, as long as the man is comfortable. I have been led to think that extreme contraction of the liver does not necessarily give rise to dropsy, so long as the general circulation is maintained by the healthy operation of the other organs. From this aspect we are again brought to see that the great risk is the blood decaying.

Experience shows that in many cases if we put the patient in bed, give him food, and moderate the use of alcohol, the dropsy disappears. Would you not cut off alcohol entirely? No. A little alcohol will make the patient a little more comfortable, and make him wish to live, and give him hope. Why should we stick him like a stone in the river of life? Further, we have to bear in mind that the liver circulation is entirely dependent on the lung circulation. We have seen this in speaking of nutmeg liver. If the blood cannot flow freely from the hepatic veins the liver-cells become choked with granular matter. Hence one of the great agents in the removal of ascites is the promotion of vigorous inspiration and expiration, we must therefore be careful not to make the patient too uneasy, and not to cut off his alcohol if it comforts him, and thus makes his breathing easier.

When we used to regard the degeneration of the liver as the result of chronic inflammation, we used to act on that theory, and try to get the morbid material absorbed. The prevailing treatment was, "A few grains of blue pill every three or four hours." We salivated them, and it killed them. When the inflammatory theory, and with it the mercurial treatment, were abandoned, tapping the belly came to be much relied on, and it was thought that in this way the patient might be cured. This brings me to consider when tapping is necessary. If we find that the tension in the belly is making the pulse smaller, the tension must be relieved as soon as possible. The tension in the belly hinders the breathing, so that the blood cannot get through the lungs, and is cut off from the arteries, and so the pulse becomes smaller and smaller. We must remember this sign, for sometimes the nervous action may be so much weakened that the patient does not complain of shortness of breath, though about to die of failure of breathing. Moreover, be guided by the pulse, not by the quantity of fluid. Some patients are much more depressed by two or three pints of serum than others by two or three gallons, and if we delay the tapping too long, it may be too late to lead the breathing to be stronger, and the tapping does not hinder death.

When the liver is contracted, get the other organs to supplement its functions—the lungs and kidneys especially. As pulmonary function increases, it will tend to promote more and more the absorptive power of the stomach, and the general circulation will be strengthened by rest and food. We have been brought to see that persons with cirrhosis of the liver may much improve in health and live for years, dropsy entirely disappearing. But always bear in mind that the great risk is the blood decaying; therefore these patients require much rest, fresh air, and food. The blood is made by the stomach and the liver dealing with the food, and with many kinds of food; if, therefore, the liver is failing, and the blood tending to perish, change the food more and more, and give also plenty of water to carry the food on.

These views on treatment are increasingly supported by study of the conditions that lead to cirrhosis. We have said that spirit-drinking causes fibroid change in the liver, but is it true? Can abuse of alcohol produce cirrhosis, no other cause being in operation? I have seen extreme cirrhosis of the liver in a boy of fifteen—it was said he was given to drink, but he had not had time to drink much. On the other hand, it is astonishing to see sometimes what enormous quantities a man will drink for many years, and yet get very little cirrhotic change in the liver. I very much doubt if the mere abuse of alcohol can give rise to cirrhosis.

Let Nature guide you. We notice this in Nature, that as soon as the sun begins to operate on this earth, there is more performance. The leaves open out, the earth steams, the birds sing, human beings get up and go to work, everything begins to perform. All functions are called into increasing activity by the sun. As the sun gets higher and hotter, there is more and more activity in water, the leaves transpire, the men sweat. If it is going healthy there is more and more cheerfulness and brightness. The man gets up, and on his way to his work goes whistling along. All this healthy and happy activity means increasing circulation. Look at clear serum, or at fresh-drawn blood, and see how bright and beautiful they are. For a tissue to go on in its healthy nutrition it must have power of circulation and cheerfulness and ease. To work easy is to be healthy.

If a man takes too much alcohol it depresses and depresses, it makes feeling gloomier, it lessens oxidation, it slows circulation. We give alcohol to prevent waste—that is why it is so useful in phthisis, but that is why the abuse of alcohol hinders oxidation, slows circulation, and destroys healthy feeling.

With the loss of healthy feeling appetite fails, and here we approach the cause of cirrhosis. It is not alone the taking of alcohol, but the loss of food. The great function of the liver is to work food up into living plasma, and living plasma up into blood. With the loss of food this function is less and less excited, and with the excess of alcohol there is more and more hindrance to oxidation—now as the function fails the part wastes (Darwin). The wasting leads to contraction, but there is more than contraction, there is growth. The loss of function tends to weaken more and more the serous circulation through the connective tissue of the liver, there is increasing stagnation, and the nuclei are swelled up; these nuclei grow to spindle cells which become more and more packed together. It is a similar process to what occurs in fibroid change in all parts of the body.

If you go and talk to horticulturists who have to study how to promote healthy nutrition in plants, they will tell you, "It does not do to leave herbaceous plants too long in this place or in that—they get woody." They get woody, they get stiff, and the circulation cannot go on through them; they stiffen and they rot. It is just so with animals; they must have no too narrowing conditions.

Too much restriction, too much hindering of function, tends to bring about fatal fibrosity.

LECTURE XLVI.

CANCER OF THE LIVER.

Cancer of the liver is very rarely a primary affection. To speak from my own experience, it results from antecedent cancer growth in other organs, and it may occur consequent to cancer of any part of the body. There are, however, two distinct classes of cases of cancer of the liver; in the first, the cancer begins somewhere in close proximity to the liver, and invades it by extension of the original growth; in the second the liver is not affected by direct extension, but it contains scattered secondary growths. Cancer invading the liver may begin along the lesser curvature, or near the head of the pancreas, or in the gall-bladder. The scattered secondary masses are most frequently found in connection with primary cancer of some part of the gastro-intestinal tract, but may be secondary to cancer of the uterus, the testicle, or any other part.

The cases in which cancer growth invades the liver may again be divided into two classes, first, those in which the common duct is not completely blocked, so that jaundice is absent or slight; and, secondly, those in which the duct is quite impervious.

If cancer grows into the liver from the lesser curvature of the stomach, there is no jaundice. These are cases of medullary cancer, and on post-mortem examination we see a moderately firm, grey mass, more or less nodular, and very vascular, which has destroyed much of the lesser curvature of the stomach, and has extended to the under surface of the liver. The symptoms are mostly gastric pain, wasting, and vomiting, occurring in middle-aged or elderly perons; I never remember to have seen cancer of this kind in young subjects, who suffer more frequently from peritoneal cancer.

Scirrhous cancer may begin in the wall of the gall-bladder, and extend up into the liver-substance. In this condition, also, the common bile-duct may escape, so that there is no jaundice. A tumour is sooner or later felt, and the liver grows larger and harder.

Where the cancer is about the head of the pancreas (by this we mean simply, that the growth is in this region, for we do not know exactly where it begins), we find at the post-mortem examination, a mass of new growth, similar to cancer of the lesser curvature of the stomach, but commonly firmer, it contains more fibrous tissue, and more approaches scirrhus in consistency. The growth extends up to the second portion of the duodenum, involving the orifice of the common bile-duct, and completely blocking it. The liver is not in these cases necessarily involved either by primary or secondary growth, but as there is always hepatic enlargement, and the symptoms are mainly referable to hepatic disturbance, it is usual to speak of these cases as "cancer of the liver." We find the upper portion of the bile-duct above the mass extremely dilated, the cystic duct somewhat dilated, and the gall-bladder dilated and charged with concentrated bile. The concentration leads to concretion, and it is therefore not uncommon to find a number of black gall-stones of recent formation. The association between gall-stones and cancer is so common, that you may be asked, "Do you think that gall-stones give rise to cancer?" I want you to answer, "Gall-stones are a common result of cancer occluding the common bile-duct." The dilatation of the bile-ducts extends into the liver, so that it becomes enlarged, and during life the edge may be felt below the ribs; the enlargement being uniform, the surface is smooth. The colour of the liver is very striking, it is no longer red, but olive-green or bronze, and from the cut surface bile everywhere oozes; the liver seems to consist of a mass of small tubes, which are the dilated bile-ducts. There may be secondary cancer scattered in other organs, especially in the lungs, and we find the whole body stained with bile.

What is the suffering in such cases? At the onset the symptoms are extremely obscure. The patient has a vague uneasiness, which he cannot describe in words, in the upper part of the belly. "It is not pain," he says, "but I am so uncomfortable." Great gloominess is a marked feature. You will very likely hear the patient's friends say that he is "hipped." There is loss of flesh and increasing weakness, and we hear the friends make a familiar remark, "Don't you think he is breaking up?" At this stage you are likely to be asked, "Do you think it is anything serious?" Answer, "Yes." We must be serious about it, and recognise that if anything is to be done, it must be done at that stage. Weakness and sleeplessness are more and more complained of, the disturbance at night being often due to pain, consequent on the stretching of the parts. Soon there are signs of pressure.

In some cases the growth is more towards the portal vein than towards the duct, and ascites is the marked symptom. Sometimes the duct is more involved, and jaundice attracts our attention. Sooner or later there are commonly both. Where there is much ascites you are likely to be asked, "Do you think it is cirrhosis of the liver?"

When jaundice come on we often say to ourselves that there is no evidence of gall-stone, and that the jaundice is too persistent for simple catarrhal jaundice, which usually disappears in a week or two, while this persists week after week, and there is increasing evidence of constitutional failure.

On the other hand, the symptoms may closely resemble those of gall-stone. We can have little doubt that in such cases the growth presses on the duct before it completely blocks it, and thus gives rise to some distension of the gall-ducts, and owing to the distension spasmodic efforts at expulsion are made from time to time. Then we get severe pain and vomiting, in association with jaundice, and dilatation of the liver. These spasms may be repeated at intervals, and the jaundice in some degree varies. In such cases we can only say, "There is evidence that the common duct is partially blocked, and the liver somewhat enlarged, but we cannot yet give a definite opinion as to the cause of the obstruction—time will reveal whether the obstruction is due to something which will go on from bad to worse until it kills." Why want to look too far into the future? If we say it is cancer, all hope will be shut out from the patient's mind. Nature's ways are the best, and so wait until she reveals the certainty.

As time goes on the urine becomes more and more charged with bile, the fæces have persistently lost the appearance of bile, and the colour of the jaundice becomes deeper, darker, with more tint of dark green. This points to cancer. The blood is perishing more and more, pigment is accumulating, and the liver-cells no longer form a healthy yellow bile. We do not see so dark a tint in jaundice due to gall-stones. All this time no mass may be felt in the abdomen to indicate a morbid growth. It is, however, beyond question that there is permanent stoppage in the common duct, and it is evidently something malignant, for it is destroying the patient's health more and more, and cannot be arrested. The liver continues to enlarge, but the surface may remain smooth, and the liver not be exceedingly hard. Nutrition fails more and more owing to the destruction of the blood. There is ever weaker circulation, and loss of cerebro-spinal power, less and less food is taken, and the patient dies of exhaustion. Remember that in some cases symptoms like those of acute atrophy may precede death.

In cases in which there is ascites, it precisely resembles that which occurs in cirrhosis of the liver, and if there is no jaundice the distinction is commonly difficult. In such cases the liver is not enlarged, but we are usually guided by the pain, and by the more rapid failure of health in cases of cancer. But never be surprised if you are exceedingly bothered in distinguishing between cancer compressing the portal vein, contraction of the liver from syphilis, and contraction of the liver from cirrhosis. A patient was admitted to the hospital with ascites, and after the fluid had been drawn off, what were thought to be cancerous nodules in the liver were felt, but at the post-mortem examination I found syphilitic disease.

In secondary cancer the liver becomes enormously enlarged; no other condition leads to such great enlargement. There are many masses in the liver, of all sizes, very circumscribed, standing out in striking contrast to the liver substance. These areas have an elevated rounded edge, and are very vascular, but the more central portions are less vascular, and are depressed. The active growth of the cancer takes place at the vascular edge; the depressed part is paler, commonly yellow, and in some cases exceedingly yellow. This "umbilication" means complete death in the centre of the mass. Sometimes we find the dead centre has crumbled away to form a cavity. This condition of cancer usually ends in a few months. It may indeed go on for two years, but generally the case terminates within the first year.

When cancer supervenes in the liver in this manner, usually the first condition noticed is enlargement of the liver. There may at first be no nodules at the surface, which is then found to be smooth, making the case very perplexing. We must be careful not to give too positive an opinion; it is a very serious thing to say it is cancer of the liver, and we must not do so until there is no longer possibility of doubt. Usually, however, we find evidence of cancer in some other part of the body, and in course of time we notice that the enlargement of the liver is increasing, and that the failure of strength is more marked; there is generally also persistent pain or uneasiness about the region of the liver, but usually no jaundice. After a while we notice that the surface of the liver is no longer smooth, but nodular, and the liver is felt to be firmer and harder. Then we have no doubt that there is cancer.

It is surprising how such patients struggle for life. They will continue to walk about with the liver enormously enlarged and nodular. There is not the excessive failure of liver function that we see when the duct is blocked. The body is not so poisoned, and there is more healthy feeling, so that life is much more bearable.

An effort has been made to kill cancer by injections, to destroy the whole growth. But Nature's way is to block it up by throwing out inflammatory material, and to jam it up in fibroid growth. I am impressed that there is a natural attempt to arrest cancer, to cure it. But other organs are secondarily involved, and other functions are hindered, and the patient is killed.

LECTURE XLVII.

DISEASES OF THE SPLEEN.

The most common morbid change in the spleen is simple congestion, passing on to œdema, and leading to swelling. It is very commonly met with in some degree in death from all forms of fever. In saying all forms of fever, I want you to understand, not only the specific fevers, but all other febrile conditions. The morbid appearances of congestion and œdema of the spleen are as follows:—The organ is much enlarged, being often two or three times its usual size, and is consequently much heavier than normal. It is soft, so that its substance breaks down when touched. On squeezing the spleen a thin bloody fluid escapes. Owing to the great accumulation of blood, the tissue is much stretched, and very commonly there are hæmorrhages into the substance of the spleen. The lymph cells are often much increased in number.

This enlargement is common in specific fevers, and notably in typhoid fever, in which the enlargement may often be felt in the living subject. After death we find the organ swelled and soft, but, if it has been enlarged for two or three weeks, it is firmer, and is to be felt with a distinct edge during life. A very soft, congested spleen is only to be met with after very rapid death from fever. In typhus fever the spleen is very soft, and usually almost black. These are the softest spleens we meet with, and they are black from death in the blood: there is much pigment and much hæmorrhage. The enlargement of the spleen is usually considerable in typhus fever. It has been stated that the spleen has been found to be ruptured in typhus fever, but I am disposed to think that in these cases the spleen has been ruptured in removing it from the body. It is so soft that it breaks at a touch, and I have been able to find no satisfactory evidence that rupture occurs during life.

There is another fever in which great congestion of the spleen occurs, this is ague. In consequence of repeated attacks of congestion, the spleen becomes swollen. The congestion occurs in each attack of shivering, affecting the lungs and the liver, as well as the spleen. The repeated congestion extending over weeks and weeks, leads to exudation and hæmorrhage, so that the spleen becomes swelled, and firmer than normal. If aguish attacks occur during many months, the spleen becomes permanently hypertrophied and much firmer than normal.

We are sometimes puzzled by finding the spleen in this condition, and we suspect ague because the patient has lived in an ague district, but there is no history of definite attacks of ague. Is it possible for a patient to suffer from ague-spleen, and from other malarial trouble, without having ever had well-marked paroxysmal attacks? Experience has shown us that this is so.

A patient comes to us with enlarged spleen, and no other symptom of disease. Is it ague? we wonder. He tells us that he has never been out of London, but does that make it certain that he has never suffered from ague? No, for now and then aguish attacks occur in people who have never been out of the City. I saw one case in which there seemed to be no doubt that the disease had been contracted while living in Long Acre. We must bear these facts in mind, or when a patient comes to us with an enlarged spleen, we may be unduly troubled by thinking that he is suffering from the onset of a fatal disease. We must remember that it may be a malarious condition, and not leucocythæmia.

There is another form of congestion of the spleen, the so-called heart-spleen. This spleen is not soft, but firm. What are the appearances of the spleen met with in the bodies of patients who have died of heart disease? Very commonly the spleen is smaller than normal, firmer than normal, and has a very distinct edge. When grasped in the hand it is evident that the tissue is much condensed. When we cut into it we feel that it cuts more firmly than normal, and we see that it has a dark venously congested appearance. Why is the spleen firmer than normal? The hardness is due to the same causes that produce red induration of the lungs in mitral disease, because, owing to the great venous congestion, the water is by degrees displaced from the tissues by accumulation of albuminous matter.

Embolism of the spleen is common in heart disease. What are the appearances of the spleen when it has undergone embolic changes? If the emboli are small, there is not usually much enlargement. The spleen is not usually much darker than normal, and if we cut into it we find, in cases in which the changes are very recent, dating from only a few hours or days, merely black patches in the spleen—hæmorrhages. The next change that occurs is, that the extravasated blood becomes decolorised, the black dead blood that has torn the splenic substance turns of a rusty red colour. Over these rusty red patches there is a noticeable depression on the surface of the spleen, and there is a distinct line of demarcation between the patch of embolic change and the normal splenic tissue. The patches of embolic change are nearly always wedge-shaped, with the base of the wedge at the capsule of the spleen, and this characteristic situation and shape enable us to recognise embolic change in all stages. If the morbid change is further advanced the patch has become yellow. The red pigment has gone, and there is evidence of reparatory change. There is lymph effused along the edge of the patch, and this lymph is in process of conversion into fibroid tissue. As time goes on the greyish-yellow substance is more and more absorbed, but the fibroid tissue remains and leaves a permanent cicatrix, and it is in this manner that emboli are cured. These cicatrices on the surface of the spleen with much depression are always left as evidence of old embolic change. The changes which take place in the kidney as a result of embolism are quite similar. To sum up, they are: first, a more or less wedge-shaped patch of hæmorrhage, with the base of the wedge at the surface of the organ. Strictly speaking, the embolic area is cone-shaped, but what we see is the section of the cone. Secondly, decolorisation of the perished blood takes place, till a greyish yellow mass remains, consisting of blood that has undergone fatty degeneration. Lastly, a fibroid cicatrix is produced.

Sometimes there are several recent emboli, and then the spleen is usually much enlarged. If, in a case of heart disease, especially rheumatic heart disease, you find that the spleen is much enlarged, suspect embolism of the spleen. If there is pain and tenderness in the splenic region further evidence of embolism is afforded, and a temperature rising in the evening and falling in the morning gives still stronger evidence of embolic change. Some of these cases with very extensive embolism pass into a typhoid condition, which has been mistaken for typhoid fever or pyæmia. Therefore, in every case of enlarged spleen examine carefully for valvular disease of the heart, and in heart disease do not forget the possibility of embolism of the spleen. May mistakes be made, even when reasonable care is taken? Certainly, for I have known a case watched in the wards for weeks and weeks, and believed to be typhoid fever, turn out on post-mortem examination to be one of endocarditis.

A large, firm, hypertrophied spleen, due to protracted congestion, occurs as a consequence of cirrhosis of the liver. It is a large, firm, heavy spleen, with a reddish appearance. Niemeyer speaks of enlargement of the spleen as one sign of cirrhosis of the liver, and leaves the impression on one's mind that enlargement of the spleen invariably follows cirrhosis of the liver. He argues a little too much from theoretical considerations, and, practically speaking, the spleen is not always enlarged in cirrhosis of the liver. Why is this? Probably because the tissues of the spleen were much atrophied and were unable to swell, or else the swelling was prevented by thickening of the capsule. You should remember, apart from theory, that the spleen is not always enlarged in cirrhosis.

In leucocythæmia we find the largest spleens met with. The spleen reaches as low as, or lower than, the umbilicus, stretching obliquely across the abdomen until it may pass the middle line, and along the inner margin we feel a distinct notch in the anterior border. The edge is very distinct, and is thick and rounded. On firm pressure there is great resistance, showing that the spleen is much firmer than normal. The patient is not extremely anæmic; the skin has not the extremely white appearance seen in Hodgkin's disease, but has rather a yellow colour, that might be confounded with jaundice; it is like the yellow colour seen in pyæmia and in other conditions in which blood is dying; the lips are not pale, but usually have a bluish-red colour; the belly looks extremely big because the spleen is so large, but the rest of the body is wasted, the muscle being poorly nourished; the face has a drawn, anxious appearance, such as is seen whenever the belly is much stretched, and it is known as the "abdominal expression."

These patients come complaining of weakness and of shortness of breath, and of loss of flesh, and they say that the belly feels tight, but they especially speak of the increasing weakness. Whenever the colour-properties of the blood are being destroyed, breathlessness and muscular and nervous failure come on. It is only a question of time, until the heart-muscle fails to such an extent that the circulation cannot go on. The blood under the microscope seems to contain only white corpuscles—we can see no red ones. There really are very few red corpuscles, and these are the carriers of the colour-properties of the blood. Do not speak of it as a colouring matter. There is no clearness of thought about that idea of a dye; it is much better to speak of the colour-properties of the blood. On examining the abdomen, we feel the enormously enlarged spleen, with the characters I have described; and, in addition, I should mention that the surface of the spleen is very smooth, not nodulated, as in Hodgkin's disease.

With rest the patient may recover strength in some degree, but this is only very temporary, and he soon begins to go down hill again. These patients are liable to feverish outbursts, as in lymphadenoma; but there is another symptom that is very characteristic of leucocythæmia. As the muscles of the body waste the heart-muscle wastes, and also the muscles of the vessels, so that they are liable to rupture and hæmorrhage to occur. It may be severe epistaxis, or it may be bleeding from the gums, or purpuric spots may form in the skin. But even after the hæmorrhages the patient may recover strength for a time; but the enlargement of the spleen persists, and so does the condition of the blood. The spleen may extend almost down into the pelvis. The liver also commonly becomes somewhat enlarged, but its surface remains quite smooth. We may also find enlarged glands in the neck. The duration of the disease may be from several months to one or two years. Death may take place from hæmorrhage—from the nose, the lungs, or the bowel; or the repeated feverish outbursts may lead to death from asthenia.

What is the appearance of the spleen after death from leucocythæmia? The spleen is enormously enlarged, and is of a red colour; we do not see the suetty masses found in lymphadenoma; it usually weighs several pounds; it is firm, and the red shows varying shades of intensity, some parts being so deeply red that we think that hæmorrhage has occurred in the indurated spleen. Under the microscope it seems to be simply an enormous hypertrophy of the spleen, the cellular and fibrous elements being extremely in excess. The enlargement of the liver and of the lymphatic glands seems to be a similar simple hypertrophy. In cases in which the patient has died with purpuric symptoms there may be widely scattered hæmorrhages.

The antecedents of this disease are unknown. Most of the cases I have met with have been in young adults, and more commonly males.

In Hodgkin's disease the spleen is often enlarged, and the liver and lymphatic glands are affected; and hence this disease has often been confounded with leucocythæmia, but the two conditions are essentially distinct. We never see in Hodgkin's disease such an enormous increase of white corpuscles; and, on the other hand, there is usually extreme pallor, which is not a symptom of leucocythæmia. Although the glands swell in both diseases, yet in leucocythæmia they are never affected to such a degree as in Hodgkin's disease, in which the main stress falls on the lymphatic glands. Further, in the spleen in Hodgkin's disease the morbid change occurs in patches, instead of a uniform overgrowth, as in leucocythæmia. We find in the spleen in Hodgkin's disease a number of grey masses, very much of the colour and consistence of cut parsnip, varying in size from that of tubercles up to that of large walnuts, and associated with similar growths in the liver and lymphatic glands.

Tubercle in the spleen is usually associated with strumous tubercle in other organs. In the spleen there are yellow masses of caseous tubercle, softened in the centre. Such tubercular change in the spleen is much commoner in children than in adults.

We may find a lardaceous spleen when the rest of the body is free from lardaceous change, showing that the spleen may be the first organ affected in lardaceous disease. There are two forms of lardaceous spleen. In the first there is a uniform lardaceous infiltration throughout the entire organ. The spleen cuts like suet, and is infiltrated by a dull grey material. On treating the cut surface with iodine, it changes in colour to a deep mahogany brown. In the other form the deposit is in little spherical masses, which give the cut surface an appearance as if sago-grains were imbedded in it, and the condition has hence been called "sago-spleen." When we add iodine the little masses turn brown. Commonly the liver, the kidneys, and the mucous membrane of the ileum are also lardaceous. With the microscope we see a dull, cloudy, grey, hyaline appearance; the tissue is infiltrated with a comparatively formless granular material. The deposit is mostly limited to the capillaries and small arteries, and thus these have rather a sharp outline. What is the lardaceous change? At one time, owing to the iodine reaction, it was thought to be a starchy material, and it was spoken of as the amyloid disease, but it is now known that the infiltration is with an albuminous material. The lardaceous change commonly takes place in diseases in which there is a slow decay in the blood, hence it is met with in phthisis and syphilis, in bone disease of long standing, and in protracted suppuration.

Syphilitic disease of the spleen is very rare, but gummous change, like that which affects the liver, the testis, and other organs, occasionally occurs.

Cancer only affects the spleen by extension from other organs.

Now and then we meet with a large blood-cyst in the spleen. The origin of these cysts is uncertain, but a blood-cyst might arise from a ruptured aneurysm.

Fibroid change occurs in the spleen in connection with fibroid change in the kidney, or granular contracted kidney. The vessels are thickened, there is a great excess of the connective-tissue element, and the organ is much diminished in size. I know of no symptoms referable to the splenic change, except that the skin may be yellowish, and the patient may become more weak and breathless, showing signs of blood degeneration, not improbably connected with the fibroid change in the spleen.

We may meet with a firm enlarged spleen, in which there is a simple increase in the normal structures, and no cause can be traced. In such cases it is probable that the hypertrophy is the remains of protracted venous congestion.

LECTURE XLVIII.

ENDOCARDITIS.

The morbid appearances of endocarditis are in the early stage very similar to those of inflammation of a serous membrane, Inflammation most commonly affects the valves of the left side of the heart, and especially the mitral valve. The first thing that we notice is that the valves have lost their bright appearance, and have become dulled and opaque—the tissues of the valve are swelled up by a fine granular material and much softened. Lymph may have been poured out on the surface of the valve, and have collected in small granular masses of about the size of a pin's head, and these may become aggregated to form larger masses. We call them vegetations. They may be limited entirely to the edge of the valve, or they may be collected in such masses as almost to close the orifice. In such a case on separating the vegetations we find the valve much destroyed. If it is the mitral valve, we may find the chordæ tendineæ softened, and perhaps one or more of them ruptured, with a little lymph attached to the torn ends, proving that the rupture was due to inflammation. Such destructive inflammation of a valve is called ulcerative endocarditis.

On microscopical examination we find that the surface of the vegetations consists of a fibrinous granular material. Nearer to the valve we notice quantities of leucocytes. Still nearer these cells are evidently enlarged, or at least we find quantities of larger spherical cells. In the tissue of the valve itself we see spindle cells. We think that if the patient had lived long enough these spindle cells would have led to much thickening of the valve.

Inflammation may affect the lining membrane of the heart away from the valves. It not unfrequently begins in the endocardium of the left auricle about an inch above the root of the mitral valve, and from here we think that it may take days in travelling to the valve. It may also occur in the lining membrane of the left ventricle. Ulcerative endocarditis, septic endocarditis, diphtheritic endocarditis (it is known by all these names), may occur away from the valves. It sometimes begins in the septum between the ventricles, and it may rapidly perforate this structure. It has been called septic endocarditis because it is such an extremely poisonous condition, and may lead to death with only a single small diseased spot in the body. It occurs usually in young subjects, fairly nourished, and in the most mysterious conditions. A patient may be going about, and suddenly be taken with signs of rapid failure of the heart, and die of this disease. I think that it is most probably due to some extreme nerve-failure.

When we find on post-mortem examination that there has been recent acute endocarditis, we usually find evidence that it is a secondary affection, that there is recent acute inflammation grafted upon old disease of the valves. We learn that fatal acute endocarditis is most commonly a result of old disease. This old disease may either have been fibroid thickening of the valve, the sequel of former attacks of acute endocarditis, or it may have been atheromatous degeneration of the valve. We have then three things to consider, first, atheroma of the valves; secondly, the conditions which may give rise to acute endocarditis in a previously healthy valve; and thirdly, the results of acute endocarditis, and especially the process of fibroid thickening and contraction of the valves.

Atheromatous disease of the valves is due to excessive and destructive tension, and affects especially the aortic valves; we therefore meet with it chiefly in the bodies of men, and after middle age—that is, after immense wear and tear; the view that this disease is due to repeated and excessive tension is corroborated by certain exceptional cases in which we find this change in the bodies of young men. I call to mind the case of a young man who had been employed in carrying bricks up high ladders, and he died of atheromatous valvular disease. The post-mortem examination left a clear impression on my mind, that the origin of his disease had been the excessive strain to which his heart was exposed by his employment. We are moreover led to attribute atheromatous change to excessive tension because it usually occurs in other parts of the body in which it is obvious that there must be undue tension at times—we see it at the orifices of vessels, in the sinuses of Valsalva, and in those vessels which have to bear the greatest pressure. Again, the only condition in which atheroma occurs in the pulmonary artery is when the artery has been subjected to extreme tension, consequent on fibroid or other contraction of the lung.

Atheromatous disease of the aortic valves generally begins about the sinuses of Valsalva, and extends into the substance of the valves themselves, leading to fibroid thickening. Owing to the contraction of this fibroid material, the valves are drawn upwards and outwards and may be everted, and in this manner they are rendered incompetent to close the aortic orifice. Such valves commonly become calcareous, and the loudest and harshest bruits met with are heard in cases with this calcareous change.

In what conditions is acute endocarditis liable to affect a previously healthy valve? There is a liability to acute endocarditis in certain poisoned conditions of the system, and in thinking of these we always give the first place to rheumatic fever. By far the greater number of cases of valvular disease of the heart arise in connection with rheumatic fever. Of the manner in which acute endocarditis comes on in rheumatic fever I have spoken in lecturing on that subject. Endocarditis especially accompanies rheumatic fever occurring in young subjects. There is a proportion of cases in which we cannot attribute the valvular lesion to rheumatic fever; there is no history of that disease, and it would seem probable that the endocarditis has been connected with some other morbid state of the body. One morbid state with which acute endocarditis is undoubtedly often connected is chorea, which itself may be a rheumatic condition. I have also found vegetations indicative of endocarditis after death by pyæmia, Bright's disease, pneumonia, typhoid fever, scarlet fever, and cholera. I am disposed to think that whenever the serous circulation of the body is failing to an extreme degree, the serous circulation through the mitral valve may become so impeded that the valve swells up, and vegetations are deposited upon it. Endocarditis may also arise from injury, from a blow on the chest, or from sudden severe strain in lifting a very heavy weight. It may be due to inflammation extending to the endocardium from the pericardium.

What are the results of acute endocarditis? It may soften the valve with the production of vegetations, and so cause death, the valve becoming incompetent and useless. But we have here to ask, How can the mere failure of action of a valve lead to death, so long as the muscle of the heart can circulate? It cannot do so—no failure of a valve can lead to death, so long as the muscle of the heart can circulate. How then is it that in some cases of acute endocarditis the heart's action is brought to a stop? By the extension of the inflammation from the endocardium into the connective tissue of the wall of the heart. The endocarditis leads by extension to myocarditis, the circulation through the capillaries of the heart becomes increasingly difficult, and the muscle becomes more and more softened until it can no longer contract. This is one result of endocarditis.

Another common result is permanent thickening of the valve, and in addition to this there is very commonly more or less thickening of the wall of the mitral orifice, and more or less wide-spread thickening of the endocardium in the cavities of the auricle and ventricle away from the valve. The thickening is due to the inflammatory elements, the corpuscles and the lymph, becoming converted into a tough fibroid material.

What are the appearances of rheumatic thickening of the valves? They are very characteristic. The valves of the left side of the heart are those chiefly affected, and of these the mitral, as a rule, suffers more severely than the aortic—the opposite to what we observed in atheromatous disease of the valves. The curtains of the mitral valve are usually much thickened, and commonly rendered rigid. The thickening extends to the chordæ tendineæ, and usually involves the apices of the carneæ columnæ, so that the muscular working of these structures is liable to become impaired, and as their action has much to do with the competency of the valve, this condition throws some light on the mode of production of mitral incompetence. The chordeæ tendineæ are many of them adherent, and the curtains of the valve are also more or less adherent, tending to form one fibroid and somewhat rigid mass. The wall of the mitral orifice is usually more or less thickened. That thickening process commonly extends in some degree up to the nearest cusp of the aortic valve. If the aortic valve is much affected, we find a ring of thickening round the attached border of the valve, communicating with the fibroid tissue in the mitral valve; the semi-lunar segments of the aortic valve have become thickened by extension of the growth from the attached border towards the free edge; as this takes place the three curtains of the aortic valve become more or less adherent. After a time the fibroid material contracts, shortening the valve, and so leading to incompetency. In such a case there may have been mitral regurgitation for months or years, until at last the aortic valve contracts in the manner I have described, so that a condition of aortic regurgitation is superadded.

In another class of cases the disease is extremely marked in the wall of the mitral orifice. In such cases there is more or less thickening of the valve, but the contraction of the dense fibroid material in the wall of the orifice is out of all proportion to the thickening of the valve. These are the cases of contracted mitral orifice with very marked præsystolic, but little or no systolic, murmur. The mitral orifice may be so contracted that it looks like a button-hole, and it is commonly spoken of as "button-hole mitral." In course of time the thickened valve may become calcareous, leading to extreme rigidity.

In most of these cases there is a history of rheumatic fever, and we are warranted in regarding these changes as the result of rheumatic endocarditis. In some cases, however, we can elicit no history of rheumatic fever, and we become impressed with the fact that a patient may have rheumatic endocarditis, and keep about all the while, the disease being so insidious that its presence is not recognised. What clinical evidence is there to support such a view? Now and then a patient comes to a medical man, who finds that there is gross heart disease, and yet the patient has never left work, and at most has suffered from slight rheumatic pains. In all cases of rheumatic fever, no matter how slight the rheumatic symptoms, never overlook the fact that the heart may be extremely affected. The disease may be, so to speak, spending itself on the heart. I have known a young creature die of acute rheumatic endocarditis while working at school. When Sir William Gull and I were studying the subject of rheumatic fever many years ago, we came to the conclusion that rheumatic fever always began in the heart. The heart is the largest joint in the body, and is certainly affected very early in rheumatic fever.

In all the chronic thickenings of the valves, endocarditis is liable to supervene amongst and swell the thickening material. Such acute inflammation is common where there is chronic damage in a valve; especially it occurs if there be conditions weakening the body. We should not overlook that risk, or acute endocarditis may take us by surprise, coming on rapidly, and in a few days softening down the substance of the valves and destroying them. Therefore it is very important in all these cases, immediately we have reason to think that acute endocarditis has supervened, to take all unnecessary tension off the valves of the heart as quickly as possible; and we endeavour to do this by guiding such patients to rest in bed, and otherwise securing them rest, as called for.

LECTURE XLIX.

PERICARDITIS.

WHAT are the morbid appearances of pericarditis? As in inflammation of other serous membranes, we first notice a loss of translucency. As you investigate disease you will be continually impressed with the fact, as I have been, that there is loss of light. Therefore I never overlook a dulled appearance. The pericardium loses its translucency; it is no longer bright and silvery looking, but has a dull grey colour. There is a fine granular exudation on the surface of the membrane, and in the cavity usually a serous fluid with a large quantity of lymph floating in it.

What is the ordinary termination of simple serous pericarditis? Commonly in recovery, and in such cases there are not usually during after-life any recognisable signs of damage done to the heart. If there has been extremely severe pericarditis, may the patient so entirely recover that weeks or months afterwards there will be no sign of disease of the heart? Yes. I once saw a boy with Dr. Ramskill who had rheumatic fever and severe pericarditis, and was so bad that we thought he was dying. Months afterwards I examined him, and I could not detect a sign of disease in his heart; he had entirely recovered.

Recovery may occur with or without adhesion. Either way is common. The pericardial cavity may be almost entirely closed. The fibrous exudation may be converted into adhesions in which new vessels are formed, and the adhesions may remain comparatively harmless for many years. In other cases the fibrinous exudation leads to thickening of the pericardium. The fibroid tissue, the remains of the pericardium, may sometimes be, as in similar thickening of the pleura, an eighth of an inch or more in thickness, a dense, tough, fibroid mass, especially developed about the base of the heart. After a time the fibroid tissue may begin to perish and become calcareous. We are astonished to find these calcareous masses forming a hoop surrounding the heart. The calcareous deposit may to some extent involve the roots of the valves. Sometimes it appears to cause little disturbance.

We must always be very careful in drawing any discouraging conclusions about adhesions of the pericardium. We should not be discouraged unless there are very certain ominous signs. There may be extreme morbid change on the surface of the heart, and yet the patient may have been able to work. Once, at a post-mortem examination, when the thorax was opened I saw a great mass over the region of the heart. This mass consisted of dense fibroid tissue, and, on cutting into it, I found a cavity containing a quantity of yellow caseous material, evidently dead pus which had become caseous. "But where's the heart?" I said. We cut deeper, and found that the pericardium was extremely adherent, and that the fibroid tissue extended into the muscle of the left ventricle. That man was a rigger, and was working in the rigging of a ship when he fell and was killed. How his heart managed to move and to work with his arms and legs I do not know. We learn that as long as the heart is able to carry on the circulation and breathing, whatever sign of damage we may find in the heart, we must avoid discouraging the patient. It is evident that in these cases of pericardial adhesion, if new vessels form, and the circulation can get through the fibroid tissue, the adhesions are rendered comparatively harmless. The most adaptable thing on the face of the earth is the human heart.

When we find pus in the pericardial cavity, we have to deal with cases which have had a different origin from those of simple serous pericarditis. Suppurative pericarditis usually ends in death.

How does pericarditis kill? When I have found fatal pericarditis, I have always noticed that the muscle of the left ventricle was flaccid. This is the cause of the fatal termination —that the disease renders the muscle of the heart unable to contract. Not only do we find it flaccid, but we notice that the muscle is no longer healthy and red, but has a dirty slaty-red colour. We lose the appearance of the muscle-bundles, and see a more or less confused granular soft mass. There is inflammatory softening of the muscle, myocarditis has supervened on pericarditis. The actual contractile elements, the transversely striated muscular fibres, never undergo inflammatory changes, except in so far as they are softened and disorganised. The inflammatory elements, the corpuscles and the lymph, accumulate in the connective tissue of the heart, and the muscle is destroyed because the capillaries are compressed by the inflammatory exudation, and the blood-supply thus cut off. We recognise that there is a degree of myocarditis which must necessarily be fatal to the muscle of the heart. When pericarditis kills, it is because the inflammation has extended from the connective tissue of the pericardium along the connective tissue of the wall of the heart, softening the muscle until it can no longer contract.

How are we to estimate the degree of myocarditis during life? By the lungs becoming moreand more congested and by consequent dyspnœa. We cannot measure it by physical signs; we can only estimate it by the degree of dyspnœa. May we have pericarditis with loud friction and very little dyspnœa? Certainly; and the amount of noise is no criterion as to the danger.

What are the conditions leading to the onset of pericarditis? They are very similar to those of pleurisy, excluding phthisis. To sum them up for you shortly, they are—

(1) Conditions in which there is poison in the blood, especially rheumatic fever. The pericarditis of rheumatic fever is always sero-fibrinous, never purulent, and for this reason recovery from it is the rule. Pericarditis, with rheumatic fever, does not kill unless the heart is much damaged by previous inflammation. Other poisoned states which may give rise to pericarditis are scarlet fever and the presence of bile in the blood.

(2) A blow on the chest may produce pericarditis, either sero-fibrinous or suppurative, and it may do this without producing fracture or external wound. I may tell you, in this connection, that the heart's wall may be ruptured by violence without there being either external wound or fracture of bone.

(3) In cases in which we find pus in the pericardium, we should at once know that the case has not been one of rheumatic fever, and we should suspect pyæmia. In pyæmia there is sometimes a small abscess in the wall of the heart, and this, bursting into the pericardium, sets up suppurative pericarditis. Suppurative pericarditis is not common in pyæmia, except in connection with such an abscess in the heart-wall, but it may co-exist with pyæmic abscess in the lung. If the suppuration has not been due to pyæmia, it has most probably been a case of Bright's disease. In a case of Bright's disease in which there has been recent acute nephritis, pericarditis is very liable to come on, and such pericarditis is often suppurative. If there is no evidence of Bright's disease, search for tubercle. If, there is no tubercle, look at the bronchial glands just above the pericardium, and you may find a small abscess which has burst into the pericardium. Failing that, look for a mass of cancer lying just outside the pericardium. If you find none of these things, if there has been no injury and no operation, look for signs of Addison's disease, for the inflammation is not due to rheumatic fever. I spoke of operation. Inflammation may pass from the root of the neck along the vessels to the pericardium. There was a patient who was operated on by Mr. Maunder for aneurysm, the carotid and subclavian arteries being tied. Two or three days afterwards, as I was leaving the Hospital, the house-surgeon came to me and said, "Dr. Sutton, that man Mr. Maunder operated on is complaining of severe pain in the epigastrium. What do you think can be the cause?" I at once suspected what it was. The man died of suppurative pericarditis.

Pericarditis commonly comes on with pain in the epigastrium, over the ensiform cartilage. As the inflammation rapidly stretches the tissue of the pericardium, it may cause agonising pain, and the pain is much more severe in the early stage of the inflammation, so that in some cases the patient may die from pain at the very outset of pericarditis, even before any friction-sound can be heard. These cases are, however, very uncommon, and we cannot but connect them in our minds with an unhealthy state of the nervous system. An illustration of this is death from pericarditis in Addison's disease, which is generally regarded as a disease of nerve-tissue.

LECTURE L.

HEART DISEASE.

Always think of heart disease as heart not working easy, its rhythmical movement being very different to that of a healthy heart. If you want to know what is easy working, listen to a child's heart, and be impressed by it.

A heart may have much morbid alteration in its structure, and yet it may go so easy that the patient works and enjoys life for many years. What right have we in such instances to say that the heart is diseased?

I attended a young person for rheumatic fever and mitral affection, and the mitral bruit has continued for twenty years; yet she has been able to go about like ordinary people. What would have been the use of frightening that patient by considering her heart as diseased?

A middle-aged man once consulted me about his heart, and told me what had led him to do so. One day, after hurrying to catch a train, he felt distressed about his heart. Consequently he went to several medical men; they had all prescribed digitalis, as I saw by the prescriptions, yet I could gather no evidence of his heart working uneasily. He was able to do his work as a commercial traveller, to eat, and sleep, and feel comfortable. Therefore I could only tell him to take no medicine, and to go on as usual. How could I treat him for heart disease?—there was a mitral bruit, but I could not treat the sound.

A tall young man came into my room one day and said, "I was advised by Sir George Burrows not to marry, because I have rheumatic disease of the heart." "For how long did he tell you you were not to marry?" "He said, not for some years." "And that's some years ago now—are you to go on like this for an interminable period, not marrying because you have heart disease?" "That's what I want you to tell me," said he.

Now what was I to do? I knew him to be excitable, nervous, extremely conscious—I was going to say, devilish clever. What chance, I thought, has this poor heart? What is the best course to keep up the circulation? Shall I give him Fear, and let him hug it to his breast for years and years—never to marry because he has heart disease?

There was no dilatation of the heart, and no difficulty of breathing, and I said, "Marry." He married, and he had one child after another, and no harm came of it.

Then I was called down into Hampshire to see him one time when he went off his head. I didn't think much of it. He got well, and he wrote me a letter saying, "Tell me by all the powers why you trusted me, at a time when I neither trusted myself nor you?" The answer was simple. I neither trusted nor thought about myself or him, I only thought about the circulation of his heart. He's well now.

Aortic regurgitation is a condition that impresses our minds gloomily, yet I remember a man who came backwards and forwards to see me at the hospital for about ten years with that condition. Contraction of the mitral orifice is an extreme morbid change; yet it is wonderful how many years people live with undoubted signs of mitral contraction.

On the other hand, a heart may be rhythmically much "diseased," whilst its cavities and valves present no indications of structural morbid change. Disturbance in the nervous system may very much disorder the rhythm of the heart. For these reasons, whenever we think of heart, we should always think of it in this manner: nervous power working in the heart; light and heat, air and water-power, albuminous and saline powers in the on-flowing blood, life in the blood, enabling the heart to go easy. Many people who come to a medical man about their hearts, suspecting heart disease, want food; others want more water and air, because they have to work hard, and these elementary powers are urgently needed to ease the heart.

Structural disease of the heart is best studied from two points of view—valves and cavities. In my last lecture I described the origin and progress of morbid alterations in the valves, and we now have to ask what is the effect on the heart of valvular disease. Sooner or later valvular disease is followed by dilatation, and this dilatation first affects the cavity behind the damaged valve. Thus, in mitral incompetency or contraction, the left auricle dilates, and when the aortic valve is diseased, the left ventricle dilates. This dilatation progresses backwards through the cavities of the heart. Thus, in aortic valvular disease, after a time the dilatation of the left ventricle becomes so great that the mitral valve becomes relatively incompetent, and the left auricle dilates; there ensues increasing difficulty in the flow of blood through the lungs, and eventually the right ventricle dilates, and this is followed in due course by dilatation of the right auricle.

The valves of the heart may be diseased for many years, but this structural alteration is of very little consequence to the patient, and he is able to go about much as usual, so long as there is no marked dilatation of the cavities. Valvular disease of the heart becomes dangerous in proportion to the degree of dilatation of the cavities, and our efforts in the treatment of valvular affection must be directed to the prevention of such dilatation. Let us then ask ourselves, what it is in these cases which leads to dilatation of the heart.

In post-mortem examinations after death from mitral disease, I have often shown you how little muscle remains in the wall of the dilated left auricle, there is only a grey gristly tissue. We are led to think that the dilatation of the auricle has resulted, less from the mere failure of the valve, than from the extension of the fibroid thickening along the connective tissue of the wall, leading to the destruction of the capillaries, and to the gradual weakening and atrophy of the muscle from mal-nutrition. When a heart lies on the post-mortem table, we see the auricle, so-called, its form is there. It is then clear that the heart's motion does not depend on mere form, for there lies the heart, and it does not contract. Where does the power producing contraction come from? What is contraction? Compare it with other great workings, that of the steam-engine, and the like, and you will find that they are all due to the circulation of living energy through the formed fixed material. The contraction of the heart is due to the energy supplied by the circulating blood, and it is wonderful what these diseased hearts can do so long as the circulation through them is maintained. Though I have not yet had time to search sufficiently and to demonstrate it in the case of the left ventricle, yet I have been led to think that, in this case also, the condition of dilatation which sooner or later follows aortic valvular disease, is more dependent on fibroid changes in the heart-muscle, interfering with the circulation through it, than on the valvular deformity. It must be remembered that death from acute endocarditis and from pericarditis is often due, not to the inflammation of the endocardium or pericardium, as the case may be, but to the associated myocarditis, and that dilatation of the heart sometimes comes on rapidly after rheumatic fever in cases in which valvular affection is absent or slight, these constitute some of the facts of clinical pathological experience which have led me to the above conclusion. Therefore, in treating rheumatic heart disease, do not think of the rigid, damaged, hindered valve, but of getting power through the wall of the heart, and remember that what tends to hinder this is the contraction of the fibroid material amongst the capillaries.

We now pass on to study heart disease from the other point of view, the affection of the cavities, and this is the more instructive aspect.

At the outset, I may remark that it is almost impossible to conceive of simple dilatation of one of the cavities of the heart, since any dilatation must be due to exposure of the muscle to undue tension, to a tension leading to undue activity, and, consequent hyperæmia and growth. Therefore, dilatation is always associated with some degree of hypertrophy, and when we speak of simple dilatation we really mean dilatation with but little hypertrophy. When we speak of a dilated hypertrophied left ventricle, we commonly mean that the hypertrophy is well marked. As the left ventricle dilates, the muscle tends, of course, by stretching, to become much thinner than normal, but at post-mortem examinations we never find the wall of the left ventricle, however dilated, much thinner than normal, except at the extreme apex. It is evident, if in a case in which the cavity is much dilated, we find the wall of about a normal thickness, that there must be considerable hypertrophy of the muscle, or the stretched wall would have appeared thin. That is what I should term a moderate degree of hypertrophy. When we speak of dilatation and hypertrophy of the left ventricle, however, we commonly mean that the wall of the dilated cavity looks much thicker than the normal wall; it may be an inch, instead of half that thickness. Cases of dilatation and hypertrophy of the several cavities of the heart fall into two classes, according as they are or are not associated with valvular deformity.

What are the causes of dilatation and hypertrophy of the left ventricle? One of the most common conditions giving rise to these changes is disease of the aortic valves. This arises, as I have already told you, from fibroid thickening, consequent on acute endocarditis, rheumatic or other, and from atheromatous and calcareous changes. In rare cases the valvular deformity is a result of syphilis. Aortic obstruction and regurgitation are usually associated, but one or other condition may greatly predominate. In cases in which the signs are those of extreme obstruction, whilst the signs of regurgitation are trifling or inappreciable, the cause is atheroma, with calcareous degeneration, the valves being converted into rigid masses which project into the channel of the aorta and obstruct the orifice. In such cases a large heaving impulse is felt under and to the left of the left nipple, denoting hypertrophy and dilatation, with a loud, harsh, systolic bruit carried along the aorta, denoting obstruction, whilst the radial pulse is exceedingly small, manifesting that there is a diminished flow of blood into the arteries, this also revealing that the aortic valvular disease is extremely obstructive.

The obstruction may not be in the aortic orifice, but between this and the origin of the large vessels; this is generally due to rheumatic inflammation in connection with pericarditis, the inflammation having extended along the outer coat of the aorta, fibroid thickening, contraction of the thickened tissue, and constriction of the vessel resulting in process of time. The proximal part of the aorta then becomes dilated, the aortic orifice becomes dilated, and ultimately regurgitation may occur. Before this stage there may be no murmur in such cases, but the heaving, dilated ventricle, with the very small pulse, tell us beyond all question that we have to do with constriction of the aorta.

You may be asked whether dilatation and hypertrophy of the left ventricle occur when the mitral valve is diseased without any affection of the aortic valve. Your answer should be negative. In very rare cases we find some degree of dilatation and hypertrophy of the left ventricle in association with much thickening of the mitral valve, with no apparent cause, but these cases are in such a small minority that it is evident that there must be some undiscovered complication. I have only seen this condition two or three times.

It is very probable that the left ventricle is always dilated in acute pericarditis, owing to some degree of inflammatory softening of the muscle, but it seems commonly to get back to its normal size as the inflammation subsides. Months or years later, when the adhesions become very firm, the ventricle may become exceedingly dilated and hypertrophied. Then the mitral valve becomes incompetent, but there is no primary valvular disease. In these cases there is in the muscle of the ventricle a fibroid substance, remaining from the inflammation, extending around the vessels, thickening them, and compressing the muscular fibres in parts, dilatation ensuing on the mal-nutrition of the muscle. Dilated left ventricle occurs also, especially in children, as a sequel of rheumatic fever without adherent pericardium, and without valvular disease. As in all cases of dilatation there is also some degree of hypertrophy, but the dilatation is greatly in excess. The mitral valve is frequently incompetent, but only in consequence of the dilatation. I have already had occasion to refer to the significance of these cases in relation to the causes of dilatation of the heart. I have been led to think that in these cases the dilatation is the result of myocarditis, an inflammation of the connective tissue of the left ventricle, but I have not verified that inference by microscopical observation.

Fatty degeneration of the heart in cases of anæmia may lead to some degree of dilatation of the ventricle; this is a condition to which attention has particularly been drawn by German observers.

Dilatation and hypertrophy of the left ventricle may be due to syphilitic changes in the wall of the heart without any primary valvular thickening. This may cause death by leading to mitral incompetence, and the other sequels of dilatation, presently to be described, or it may lead to rupture of the wall of the heart, the blood bursting into the pericardial cavity and killing the patient. This syphilitic change may take the form either of small, definite, gummous masses, one or more, in the wall of the heart, or it may take the form of fibroid bands, very similar to those produced by syphilitic change in the liver, the lung, the tongue and other parts. In some of these cases it was a gross fibroid change, fibroid masses which cut like gristle, and formed one or more lumps among the muscle. In other cases we found no such masses, but a fine thready fibroid material extending amongst the muscle. In some cases also such fibroid change was traceable to abuse of alcohol. All these varieties of gummous and fibroid change are however rare and exceptional.

There is a very large class of cases of dilatation and hypertrophy of the left ventricle occurring in middle or late life, any valvular deficiency which is present being a consequence, not a cause, of the dilatation. It is commonly spoken of as "dilated left ventricle." It is more common in males than in females.

In a very large proportion of cases this dilatation is associated with kidney disease, usually granular contracted kidney, and if we find large white kidney, it is one containing also much fibroid tissue. In some cases of kidney disease it is indispensable to perceive that the heart's condition calls for medical treatment, not that of the kidney—that the patient really is suffering from heart disease and dies of heart disease, the share kidney failure has in leading to death being mostly secondary to failure of the heart. This is proved by the condition of the lungs found after death, and by the "nutmeg liver," and the firm dark spleen. These conditions are such as are always found, in greater or less degree, after death from heart disease, and testify that death was the result of heart disease. In some such cases there may be to the naked eye very little evidence of granular degeneration of the kidney, but the microscopical examination may reveal considerable fibroid change.

In former years it was taught that this dilatation and hypertrophy of the heart was always due to the kidney change; but when it became evident that while, in some cases, there was extreme granular contraction of the kidneys, in other cases, change in these organs was only revealed on microscopical examination, it was understood that so extremely variable a cause could not account for the dilatation; and further search was made for associated morbid conditions. It was found that in these cases the larger arteries were atheromatous; I have frequently seen gross fibroid thickening along the aorta and larger arteries associated with dilatation and hypertrophy of the left ventricle, and such atheromatous degeneration was for a time thought to be the cause of the heart disease. But further inquiries made it obvious that marked atheroma might be present when there were no dilatation and hypertrophy of the left ventricle. You will be particularly impressed by seeing that the coronary arteries may be extremely atheromatous, converted into rigid calcareous tubes, and yet there may be no dilatation and hypertrophy of the left ventricle.

We were then led to look for changes in the arterioles and capillaries, and we found changes, a condition of arterio-capillary fibrosis. As time went on, we found that not only was there disease in the arterioles and capillaries, but there was disease in the connective substance of the wall of the left ventricle, a similar process of degeneration to that seen in the contracted kidney itself, and in the blood-vessels. We found, further, that, in a few cases of dilated left ventricle, the change might be mainly confined to the wall of the ventricle, but it was usually associated with changes in the smaller blood-vessels and in the kidneys. This fibroid change is like all fibroid change; it is a stiffening and hindering process, a stenosis around the vessels.

If, then, you are asked what changes have been found in the wall of the left ventricle, where the ventricle is much dilated in association with granular kidney, your answer should be, "The connective tissue of the wall of the left ventricle is thickened and stiffened, and markedly that around the arterioles and capillaries. The longitudinal striæ in the muscle-bundles are similarly stiffened, so that the muscle looks more like bristly substance than muscle-band, and the transverse striæ of the muscle are much diminished. Besides these changes there is atrophy of the muscular fibres, owing to the contraction of the fibroid material and to the thickening of the capillaries; and, by the side of these atrophied fibres, others occur which are hypertrophied."

It is here, as it is everywhere, the rule in the body—atrophy and hypertrophy side by side. As one part of the tissue fails, the other part has to take on increased function to compensate. It should always be borne in mind that, with failing function in one part there is effort in another part to supplement the weakened action—that in Nature there is thus a balance. That is particularly to be noticed in these hearts. If weakened in one part, they become stronger in another. That supplemental proceeding is the spirit of therapeutics.

What are the causes of dilatation and hypertrophy of the left auricle? I have already spoken of the tough fibroid condition in which we commonly find the wall of the dilated auricle. If the left auricle has become dilated, it is consequent on contraction of the mitral orifice, or on thickening, deformity, and incompetence of the mitral valve; or else it is consequent on extreme dilatation of the left ventricle, the valves themselves not being thickened, but, owing to the great dilatation of the ventricle, unable during systole to close the mitral orifice. Regurgitation of blood then leads to distension of the left auricle, and eventually to its dilatation.

What are the causes of dilatation and hypertrophy of the right ventricle? The most common is some permanent obstruction to the circulation through the lungs, caused by degeneration of the lung-tissue. The most frequent of these is vesicular emphysema; but it may be fibroid consolidation, the so-called cirrhosis of the lung; or it may be simple atrophy. Another condition acting similarly is extreme contraction of the pleura, and consequent compression of the lung. As the ventricle dilates, it leads to widening of the tricuspid orifice, and incompetency of the tricuspid valve.

In some cases—in fact, in many now—I have found much hypertrophy of the right ventricle, with comparatively little dilatation; but more frequently we find extreme dilatation of the cavity, with comparatively little thickening of the wall, and these are the worst cases. In cases of the former class, a tricuspid murmur is commonly heard, provided there is a sufficient degree of dilatation to render the valve incompetent; when the dilatation becomes extreme, there is no tricuspid murmur. Therefore, a tricuspid murmur is a comparatively favourable sign.

When I have seen a case with comparatively little dilatation of the right ventricle, but with much thickening of the wall, and with the circulation through the lungs much obstructed, I have asked myself, "Wouldn't it have been better if that ventricle had dilated?" A ventricle can't send the blood through the lungs because of the obstruction. It can't send it back into the auricle because of the valves. What is it to do? The blood must become stagnant in the ventricle. Probably in these cases the wall is so tough and fibroid that the ventricle cannot dilate. For these reasons we do not hesitate to take away a little blood, if we have reason to think that the blood is stagnating in the right ventricle, so that it cannot get on with its work.

Very commonly dilatation of the right ventricle is consequent on disease of the left side of the heart, which has led to dilatation of the left auricle and increased pressure in the pulmonary vessels. Whenever, therefore, we want to measure the severity of disease of the left side of the heart, we turn to the right side, and ascertain if it be dilated, because, if the blood be unable to pass freely through the left side of the heart, it sooner or later leads to excessive distension of the right side. Do not forget that fact.

Cases are seen in which no disease on the left side can be detected by physical signs; but there are signs revealing that the right side of the heart is excessively distended and dilated. There are no signs indicative of primary disease in the lungs to account for the dilatation on the right side, nor of primary disease in the pulmonary artery; so we are led to conclude that the dilatation of the right ventricle is consequent on disease of the left side of the heart, although no bruit can be heard. That experience is not uncommon in cases of extreme mitral contraction. During the last few weeks of life the characteristic præ-systolic bruit not infrequently disappears, and the only signs denoting that the heart is much diseased are, the small, soft, and irregular pulse, difficulty of breathing, and other signs of lung-congestion, and signs of great dilatation of the right side of the heart. It is not always easy for us in these cases to avoid great error. The most extreme and fatal morbid changes in the mitral orifices and valves may be overlooked, unless we recognise, and rightly interpret, the dilatation of the right ventricle and auricle.

Constriction of the pulmonary artery, giving rise to dilatation of the right ventricle, is a very rare condition. It may, however, occur as a sequel to rheumatic inflammation in a manner similar to that already described in speaking of constriction of the aorta. There was a case once in the hospital in which there was a diastolic murmur, heard only now and again, passing obliquely from the left upper costal cartilages towards the lower end of the sternum. There was much dilatation. We found in this case the pulmonary artery constricted, the first portion of the artery much dilated, and the dilatation had led to incompetence of the pulmonary valves. The obstruction and regurgitation had led to dilatation and hypertrophy of the right ventricle.

Another very rare condition leading to dilatation of the right ventricle is pressure on the pulmonary artery, aneurysmal or other.

What are the causes of dilatation and hypertrophy of the right auricle? I remember the particulars of a case in which the right auricle was extremely dilated and the venous circulation fatally obstructed, and it was produced by a strumous growth in the wall of the auricle, extending from a strumous mass in the adjacent bronchial glands. Usually, dilatation of the right auricle is consequent on dilatation of the ventricle and its attendant tricuspid regurgitation, or on contraction of the tricuspid orifice.

It is necessary that I should speak in some detail of fatty disintegration in the wall of the heart, and also of fatty growth in the wall of the heart, which is quite a different condition.

In the latter there is a growth of fat cells accumulating in the pericardium, and growing in between the muscle-bundles of the wall. It is always most marked in the right ventricle, which we find very soft, and in extreme cases the muscle of the ventricle may be difficult to detect. It is associated with great accumulation of fat elsewhere. The danger of the body becoming excessively fat is, that the fat may grow into the wall of the right heart until it becomes unable to pump the blood through the lungs. There is usually some emphysema, but may be little or no bronchitis—and yet the patient is extremely short of breath. We say to ourselves, "How is it that, with little emphysema, and no bronchitis, the patient is so breathless?" We infer that the right heart is weak from fatty growth. From time to time such patients become startlingly livid, and they suffer more and more and cold. They are more lethargic. They have less and less appetite and less and less healthy feeling. One day in one of the attacks of lividity the patient dies.

Fatty disintegration affects the left ventricle as a rule, but in rare cases the right ventricle. It is always the result of anæmia. We notice that the muscle of the heart is of a dirty drab colour, it has a grumous look, and it easily breaks down, being much softer than normal. On closer examination we see a number of zigzag lines passing across the muscle of the left ventricle, and these are usually most clearly visible on the carneæ columnæ—they are areas in which the muscle is much replaced by fat granules. Under the microscope we see that the transverse striæ of the muscle fibres have mostly disappeared, and the fibres are filled with minute granules. This degeneration comes on in anæmia from loss of blood; in anæmia without obvious cause, such as occurs in young girls; in anæmia due to cancer, in anæmia due to strumous disease—in fact, in all forms of anæmia; the great danger of anæmia is that the heart will fail fatally owing to the onset of this degeneration. If a woman has lost a great deal of blood, and in a few weeks you find her very breathless, more so than could be accounted for merely by the anæmia, suspect fatty degeneration of the heart. I have only once seen fatty disintegration of the right ventricle, and then it was associated with extreme atheroma of the pulmonary artery secondary to emphysema.

There is another condition of degeneration in the muscular tissue of the left ventricle, known as granular degeneration. The muscle becomes very soft, very granular, and unable to contract. It occurs after fevers, notably typhus and typhoid. It also commonly occurs in drunkards; we see it with especial frequency in men who have drunk large quantities of beer. In the bodies of such persons we often find this granular condition in the wall of the heart, the kidney, the liver, and other organs. The meaning of such cases seems to be that the extreme abuse of alcohol has hindered the oxidation of the muscle, until it has perished from this granular change. Similarly, in fevers there is hindered oxidation.

In some cases there may be a very localised dilatation of the left ventricle, a bulging into the pericardial cavity. There is an aneurysm of the wall of the heart. In this fibrin may be deposited, just as in ordinary aneurysm, and it may lead to death by rupture of the heart. It is doubtless due to a localised degeneration in connective tissue.

Rupture of the heart may occasionally occur without previous bulging. Sudden death occurs, and on post-mortem examination we find the pericardial cavity full of blood-clot, and a small rent in the wall of the left ventricle. This generally occurs in middle-aged or elderly persons, in whom the wall of the heart has been undergoing slow granular degeneration. We find in such cases evidence of other tissue degeneration—granular kidney, emphysema, diseased vessels, &c.

We now come to consider the signs which manifest that the heart's function is failing, the signs of weakened heart, the heart disease. These signs are in some degree met with in all forms of heart disease. One of the earliest signs is irritable action in the heart's rhythm, felt by the patient as restlessness, very commonly accompanied by sleeplessness. He wakes many times in the night, or lies awake for hours together. This sleeplessness is a great trouble. Heart's failure is also often manifested by excessive irritability in the feeling. The nervous system becomes unhealthily sensitive, and the patients are therefore beset with worries and troubles. As the beautiful harmonious rhythm in the heart's working is lost, the world becomes exceedingly worrying, because—and this it is important to remember—the rhythm of heart and brain work together, and are disordered together, and impressions from the outer world are received according to the condition of the rhythm of heart and brain. The next noticeable symptom is muscular and nervous weakness; the patient is soon tired and prone to be apprehensive. On listening to the heart we feel that the easy rhythmical process is lost, the action is more tight and constrained. Such a hindered tight beat is common in mitral disease—the heart's rhythm is no longer adaptable to the movements of the legs, arms, and other parts. That is probably one of the great causes of shortness of breath, that the heart's beat cannot adapt itself to the varying needs of the body. The heart can live in any climate; a man can go into an oven, and the heart can continue working while the skin is nearly baked; it can exist at great heights, or deep down in the earth—it is marvellously adaptable. But for this its beat must be able to alter with the altered conditions of the circulation—it is this power that is lost in disease. A young fellow came to me about his heart; there was no murmur, no perceptible structural alteration; but his heart was irregular, and he was miserable.

As heart failure is coming on, patients complain of palpitation. By that they mean that they feel the irritable beat of the heart; it may be that they say they feel it knocking against the wall of the chest; we may often in such cases feel the irritable constrained rhythm on placing the hand over the cardiac region. Palpitation is particularly distressing whenever the patient is alone, and therefore they complain so much of its occurrence at night.

Now here let me mention to you what I have gathered about rhythm. What does rhythm mean? I soon came to recognise that rhythm and easy working go together; that which works easily is rhythmical; regular repetition, and absolutely reliable repetition; therefore it is in order. In Nature, it is traceable that all motion is rhythmical. This has been ascertained of heat, light, and electricity, and it is seen in the wavy movements in tides. I see it in the energy of fire passing into water—in boiling; in the working of wind with water; in the life of plants; in the breathing of animals. Throughout Nature, rhythm and easy working go together. Paralysis is absence of rhythm. All work means rhythm; therefore a healthy, rhythmically acting heart and power of work go together; and as the heart loses its rhythm, the patient loses his power of working.

As I proceeded inquiring for rhythm, whilst endeavouring to feel my way into the rhythm of the world, I one day stood near a running brook, and waited until my senses were governed by its rhythm; then I found that I was inwardly, and in a little while outwardly, moving to it. The brook got me into tune, and away I could go. As I got into time and tune with the rhythm of the brook, my heart's and other muscular movements were toned, agreed accordingly. My senses were governed by the mighty undeviating rhythm, and my heart and limbs moved marvellously "easy," for the government of the great is the easiest government.

For, if we turn to the other, if we observe the government of the sentimental mawkishly weak, we see, what has long been recognised "the danger in works sentimental." "They attack the heart the more successfully because more cautiously." (Dr. Knox.) Disease is rampant in persons governed by such influences. The most terrible tightness and uneasiness in heart's rhythm I have ever listened to was in the heart of a person whose mind had been wrapped in spiritualism. She murmured to me, "I cannot bear it long."

On listening to the heart in disease, we frequently notice, in addition to disordered rhythms, distinctive sounds of disease—bruits so-termed. To understand the meaning of these bruits, we have to consider that the first sound of the heart is connected with the energy flowing from the muscle in systole into the on-travelling blood. It is not a definite sound until the end of the systole, when the apex beat thrusts forward the chest wall, and the mitral valve becomes tense. At first it is a low-pitched deep sound, but at the end of systole, as the energy passes into the tightening valve, the sound becomes definite, sharp, clicking; it is finite.

It is a remarkable feature in valvular destructive disease that the definite feature of the sound is lost; it is more an infinite sound. The first or second sound fades away in a blowing manner, so that its end is imperceptible. This infinite or indefinite character is consequent on too definite fixed states that have encumbered the heart's structure and working, consequent on the rigid thickening of the valves, or on the hindering dilatation of the ventricle, or of the aorta; an abuse of the definite, which has marred the normal rhythmical timekeeping action, until time cannot be kept nor required position retained. The abuse of the definite deprives the tissues and the sufferer of energy. Energy cannot accumulate where there is too little for the discharge of daily work.

We must learn to recognise the sounds of disordered rhythm which precede the occurrence of bruits, that they may warn us in time to enable us to prevent the heart getting into a fatally injured condition.

You are familiar with my paradoxical speaking, therefore you will not be so much surprised to hear me say that it is a great aid in listening to sound to be deaf. Being somewhat "deaf," I have had to listen, and not hear until the sound came and taught me; I did not paralyse myself by striving to hear. In listening to a heart, let your ear guide you, and keep out preconceived ideas. On listening in this manner in cases in which there is disease, we notice that the sounds of the heart are abnormally prolonged, abnormally pitched, abnormally intensified.

We have searchingly to consider the disorder in cardiac rhythm. There may in some cases of mitral regurgitation of rheumatic origin be no bruit, but an exceedingly irregular rhythm, and the bruit may similarly be absent in cases of mitral obstruction. We may at first fail to hear a bruit, while more careful listening may reveal its existence, but the heart's action may be so rapid and irregular that no bruit can be recognised. We may give digitalis and slow the heart's action, and then the bruit is distinctly heard; and not only that, but the breathing is much relieved, and the patient feels much stronger, showing beyond all question that regularity of rhythm, ease of breathing, and increase of strength are all closely related.

You remember what I told you about the cases of dilated left ventricle occurring in middle-aged or elderly persons, without primary valvular disease. In these cases there is alteration of rhythm without, at first, any bruit, and it is the alteration in rhythm to which we must pay attention. One of the earliest signs of disorder in the rhythm of such hearts, a sign that they are going to fail, is a heavy, laborious, pendulum-like, swinging action; in the rhythm it sounds as if it were, as it really is, connected with a stiffening process in the heart. The working is too set. Such hearts cannot dance sweetly on as does the healthy heart.

After listening to such a heart, we can understand the patient sadly saying, "I cannot do as I used to do. What is the use of living?"

After a time, as the change in the wall of the ventricle progresses, we notice further alterations in rhythm. The beat is not regularly repeated, there may be two or three hurried beats, and then slow ones. There are varieties which have been called a "hurrying," a "trotting," and a "cantering" rhythm. In some cases the first sound becomes short and sharp, so that there is little difference between it and the second, and this is familiarly spoken of as the "tic-tac" sounding heart. It indicates that the muscle is exceedingly weak. These disturbances in rhythm are so commonly met with, that, immediately we discover them, we look for further signs of dilatation of the ventricle, although there is no bruit. However, as the dilatation of the ventricle progresses, the mitral valve may become incompetent, so that a valvular bruit is audible. But in these cases, as in those of primary valvular disease, if the heart is extremely irregular, the bruit may be inaudible, until the heart's action has been slowed by digitalis. As the rhythm becomes slower and more regular, the patient's breathing becomes much easier, and he is greatly relieved.

The difficulty in using digitalis is to know how much to slow the heart. There was a patient in Charlotte Ward with heart disease, and an exceedingly irregular rhythm. It was a desperate case, the breathing was so difficult. I gave her digitalis in large doses, and the rhythm became much more regular, and less rapid. "We're slowing down the heart nicely," said the house-physician to me the next day. Her breathing was much relieved, and she expressed her gratitude to me, saying, "Oh, doctor, I feel so much better." But the difficulty was, that I did not know how much it was safe to slow that heart, and within a very few hours she suddenly died.

Sir William Gull once told me of a similar experience of his own. He saw the patient in consultation with Sir Thomas Watson, then the leading London physician. I will give you his own words, "Sir Thomas Watson and I were treating a case of dilated heart, we gave digitalis. At our next visit, we found that the heart was much more regular, and we were leaving the house very well satisfied, when suddenly the patient died."

But remember, that with a more regular rhythm of the heart, there is great relief to the breathing, and comfort to the patient.

There is, when the heart is failing, another sign of increasing disturbance in rhythm, which it is important that you should notice, for it is evidence of increasing tension in the pulmonary artery, and of impeded circulation through the lungs. The second sound, due to tension of the pulmonary valves, heard over the second left costal cartilage, is exceedingly prolonged, of higher pitch, and of greater intensity.

It may assist you in understanding the suffering when the heart is failing, if, before going farther, I describe to you the results of heart failure as we find them on post-mortem examination—the morbid anatomy of heart disease.

We find the cavities of the heart dilated, and the wall leathery and tough, especially that of the right ventricle. The muscle has a more or less œdematous appearance, and there may be small scattered hæmorrhages. The coronary veins of the heart are enormously distended with blood. On microscopical examination, we see that the muscular and connective tissue are charged with granular matter. We learn that the heart has been overloaded by the accumulation of venous blood in its wall, and that it has been choked with serum and granular material. By such accumulations the heart becomes stiffened, until at last it stiffens into death. There is commonly serous effusion into the pleural and pericardial cavities.

Turning to the lungs, we find similarly great venous congestion, proceeding into œdema, albuminoid infiltration, hæmorrhagic exudation, and consolidation. Consider the changes carefully, for those in all the organs are similar. The venous congestion results in the exudation of serum from the capillaries, swelling the tissues by œdema; the water by dergees being pushed away by the albuminoid matter accumulating in the air-cells and lung-tissue, so that the parts become abnormally coherent, leathery, and tough; further, as tension increases, red corpuscles being extravasated in the condensed tissues. Sometimes the tissue of the lung becomes so much condensed, that it nearly sinks in water. If, then, you are asked to describe the appearance of the lung after death from chronic disease of the heart, the so-called splenized or heart-lung, say, "On section the lung is of a deep red colour, it appears much more homogeneous than is natural, it feels tougher than normal, it floats badly, and it is comparatively dry." In such a lung it is common to meet with pulmonary apoplexy—we see blackish masses of blood-clot filling up the air-cells of a portion of the lung. Immediately around the hæmorrhage the lung-substance is often strikingly free from congestion, being pale and much condensed. This experience led me to see a great use in pulmonary apoplexy, to relieve the congestion of the lungs. Therefore, when there is hæmoptysis in heart disease, tell the patient that he need have no fear, and that it will be a great relief to him to cough up the blood. If we find that some portions of such a lung are much softer than the rest, and œdematous, and sink in water, we know that pneumonia has occurred amongst the toughened tissue. Of late years this heart-lung has been called in books "red induration" of the lung. By this is meant that the capillaries of the lung have become more and more dilated by back pressure, and as a result their walls have become thickened by a slow fibroid growth, and they become varicose —further, hæmorrhages occur, with resulting red and blue pigmentation. Too much stress has, however, been laid on that condition of induration of the walls of the capillaries. My experience has been, that the air-cells are choked up with albuminous granular material, and with white and red corpuscles, and in course of time this exudation may lead to some induration.

The liver is swelled and toughened and charged with albuminous material, the intra-lobular capillaries are enormously dilated and thickened, and a slight degree of cirrhosis is very common. The colour of the cut surface is very characteristic; there are dark red spots of about the size of a small pin's head, thickly scattered on a yellowish ground. This is the so-called nutmeg liver, and is always evidence that there has been prolonged venous congestion. The dark red spots are due to the congestion being most extreme in the centres of the lobules, in the immediate neighbourhood of the central veins, and in this region there are small hæmorrhagic extravasations in the tissue. The yellow appearance is due to accumulated bile in the liver-cells. What does this accumulation of bile show? That there is some obstruction to the outflow—not much, but distinct obstruction—therefore a slight degree of jaundice is common with failing heart.

We find similar changes in the stomach, the spleen, the intestines, the kidneys, and the skin (especially in the lower half of the body). The brain and cord, with the membranes, are also affected.

In consequence of the extreme venous congestion and œdema, acute inflammation is very liable to come on, and frequently therefore, at post-mortem examination in cases of heart disease, we find one or more of the following changes: pneumonia, nephritis, gastritis, and dermatitis, the two last being exceedingly common.

Hepatitis is more difficult to speak about, except in a histological sense, and from a clinical point of view we must be careful how we use that term. If you ask me, "Do we find by naked-eye examination indication of inflammation of the liver simply due to heart disease?" I answer, "No." Nor have I seen with the naked eye the changes of inflammation of the brain, but with the microscope we not uncommonly find evidence of inflammatory changes in the convolutions.

Let us now return to the consideration of the signs of failing heart. Very commonly the patient goes to the medical man complaining of indigestion, of distress about the pit of his stomach. You will find that commonly when they have disease of the stomach, patients complain of their hearts, and when they have heart disease they complain of suffering in the stomach. The patient goes on to remark that he is so much troubled with flatulence, that his food seems to lie so heavy on his stomach, and that lately he has not been sleeping so well as he used to, he may either say that he cannot get to sleep for long after he goes to bed, or else that he wakes many times through the night. There may be either diarrhœa or constipation, and from time to time windy colic. As the sleeplessness and other troubles increase, his head suffers more, and restlessness becomes a marked feature, and the patient becomes very apprehensive. A healthy circulation through the brain is always attended by a feeling of security; a failing circulation, by a feeling of insecurity. Owing to the failing circulation through the brain, the brain-tissue becomes morbidly sensitive. The subjects of it become irritable; they are startled by noises; they cannot bear people about them; in bad cases they are even disturbed by any one walking across the room. Whenever you have to deal with a case of excessive irritability, examine the heart.

With this restlessness and irritability in our patients, there is usually increasing melancholy, and a loss of interest in their surroundings. If we recognise the case at that stage and give them a few days' rest in bed, they are much relieved. It is a serious question how to get them to rest. It does not follow that they will rest if we put them to bed. A patient may have cares on his mind, or business worries, and then he will get more rest in moving about. Sometimes, in these days, we send such patients to the Alps. It needs much judgment to guide them to rest. Remember that what is wanted is, more power in the circulation.

As the heart fails still more, the urine begins to reveal it. It is scanty, high-coloured, and loaded with lithates. In other cases, in which there is excessive nervous tension, there is an excessive secretion of very pale urine.

Up to this time there may not have been much complaint about the breathing, but now the patient says, "I'm getting so easy out of breath—I get so short of breath if I move at all quickly." Before the patient begins to complain of the breathing, the medical man may have noticed that it is a little quickened. In heart disease the breathing is rather quickened than difficult, whilst in bronchitis, difficulty is the marked feature. On listening, we merely notice that the breath-sound is harsh, and from this we conclude that there is more friction as the air passes into the lung. Why should there be more friction? Because the capillaries of the lung are swelled, and stand out into the alveoli.

If the heart failure becomes still greater, there is marked tenderness in the epigastrium, and a sense of fulness, and we know that the stomach and liver are venously congested and swelled. The symptoms of indigestion become aggravated, and occasionally vomiting occurs. Vomiting may be the most prominent symptom when the heart is rapidly failing—uncontrollable vomiting of muco-serous fluid. Hence, in any patient, if there be epigastric tenderness and vomiting, we should not fail to examine the heart, for rest in bed may be the best of all remedies.

The swelling of the liver leads to pain and tenderness in the right side, due to the stretching of the capsule. The liver is usually felt below the ribs, manifestly enlarged—swelled by accumulating venous blood—and there may be some degree of jaundice. Swelling of the spleen causes pain and tenderness in the left side.

If, now, we listen to the lungs, we usually hear a little crepitation at the bases behind, due to œdema. The patient begins to complain that it is difficult for him to lie down at night. The crepitation extends upwards, and the percussion note at the base becomes dull. If we notice the expectoration, we see a frothy, watery fluid; there is simple œdema of the lungs. Such a stage is commonly recovered from by rest.

If the failure increases, the trouble in the breathing becomes more severe. The urine becomes more scanty. There is more and more restlessness and sleeplessness. At this stage œdema usually appears in the feet and extends up the legs. Œdema of the scrotum is common. The period at which œdema comes on when the heart is failing, is a variable one. In some cases œdema of the legs is much marked before there is much sign of disturbance in the stomach or in the lungs. In others, there may be very little swelling of the legs at a time when there is evidently much congestion of the lungs and stomach. These effects are probably connected with localised weakness in the coats of the vessels themselves. We must remember that there is a rhythmical action in every vessel as well as in the heart, and it is presumable that it may fail more in some vessels than in others.

Vomiting is now apt to be very troublesome. There is catarrhal inflammation of the stomach, and vomiting of more or less ropy mucus. The belly becomes a good deal distended, but the percussion note is resonant and there is no fluctuation. Always before a serous accumulation, before ascites, there is excessive flatulent distension of the belly. The gaseous circulation fails before the serous, and we can prognosticate ascites when we see the gaseous distension. In the same way we can prognosticate catarrhal inflammation of the stomach by the occurrence of excessive flatulence.

If asked, "What is the most significant indication of failing heart?" answer, "Difficulty of breathing." The patient is unable to lie down in bed; he must have his shoulders raised so that he may better use the auxiliary muscles of respiration which are increasingly called into operation. That the breathing is rendered thus difficult by the failure of the heart's contractile power and consequent congestion of the lungs, there can be no doubt, but this is not the sole cause. I used to think that the difficulty of breathing was simply due to the failing heart, but I am increasingly brought to think that there is antecedent failure in respiration, and that this is the immediate cause of the heart's failure of power. Owing to some great disturbance in the rhythm of respiration, the oxidation of the blood is insufficient, and the heart is not able to obtain from the blood its requisite supply of energy.

Even in early periods of cardiac weakness the disorder of respiratory rhythm must, to some extent, be attributable to the disease in the heart; but it is also attributable to other conditions—the body has not been kept sufficiently in association with rhythms that are continuously maintaining respiration, circulation, and life.

It follows, therefore, that patients suffering from heart's failure should be allowed to rest in a place well-ventilated and well-lighted, where the air is sufficiently moving, dry and warm, and the skin should not be covered with more than is necessary for warmth, loosely covered, to allow air to move over it and stimulate respiration; and excessive tension in the abdomen or elsewhere, which depressses feeling and respiration, should be relieved.

Nature gives relief to the difficult breathing by sleep, and it is wonderful how quickly the œdema of the lower extremities may diminish from refreshing sleep. Unconsciousness may be induced by morphia or chloroform, or such-like agents, and afford much relief—better breathing; but I have found that whilst I thus soothed the feeling, and the respiratory movements became less struggling, yet, strength, on the whole, diminished. I knew a medical man with valvular heart disease who had to go on with his practice in order to make a livelihood, and had to take morphia to enable him to bear the suffering. He continued the morphia during several months, and it afforded him much relief, but I knew all the while that it was shortening his days. His circulation became weaker and weaker, until he died.

A hot bath commonly assists persons to go to sleep. It evidently does so by rhythmically freeing sentient actions of the skin, so lulling consciousness to rest. Sponging the face with hot water has a similar effect. These are more natural ways of assisting to sleep. They lead more to the "rest in action, and action in rest," which is the working of rhythm.

I make these remarks here to impress upon you, that for relief of breathing, the feeling must be relieved, so that the mind goes freer. For at this period the patient's mind is in a very curious condition. He is so apprehensive, that we may find it difficult to get him to speak to us. In such cases, if we listen and will be taught, we may learn that old impressions come to the surface-consciousness and perplex poor self distractedly. They dream of their past childhood, of past family troubles—ghosts of the past. Such dreams, and delirium also, may prove useful, by freeing the mind from past dead impressions, and breathing may so be much relieved. To free the mind is always to relieve the breathing. These sufferers long to be out-of-doors, in association with what is going on in Nature, and to be pulled into order and strength again, and ought we not, as medical men, to promote that more than we do? We fear Nature too much, to say the least.

At this time the friends will worry both you and the patient. They will want to know when he will die. Will he die next week, or will he die in a month? Your duty will be to listen and listen, and say as little as possible. If, gentlemen, we were rowing a boat, and were coming to a very dangerous part of the stream, we should say, "We won't know much, and we won't say much, but we'll keep feeling our way inch by inch, and we won't think too much about the boat." Their only chance is to take the case like that; and therefore I say that your duty will be mainly to listen and say as little as possible. The patient will bless you for it, for he'll have been talked at and talked at until he's sick of it. Of all the troubles of the human heart, gentlemen, there is none so bad as useless sentimentality. Work is a blessing to it; but it hates useless sentimentality.

As the failing heart comes nearer to its end, we may observe, on auscultation of the chest, that the crepitant râles have much disappeared; but the breath-sounds are more markedly tubular, and the percussion-note becomes duller. The leathery condition of the lungs, spoken of as red induration, is coming on. We may ask ourselves if there is pneumonia. It is, in such cases, commonly difficult to detect. If the temperature is normal, we may consider that there is no pneumonia; while a rise of temperature, without other evident inflammation to account for it, leads us to think that probably pneumonia has supervened. At this period of heart failure the expectoration is very characteristic. It is so exceedingly sticky that if we turn the vessel upside down it does not run out; it contains very little water, much fibrin, and more or fewer red corpuscles. This expectoration tells us that the circulation through the lung is extremely hindered. The edge of the liver may be several inches below the ribs; the stomach is excessively distended with gas; there is ascites; the subcutaneous œdema is more general, the back commonly becomes very œdematous, and the hands and arms are swelled. As the venous circulation through the brain becomes more obstructed, stupor tends to come on.

As the heart failure becomes extreme, the arteries of the brain become less and less filled, whilst its veins are ever more and more choked with blood. Moreover, in these conditions of heart-failure the red corpuscles—the breathing organs of the blood—tend largely to perish, so that in this way also the oxidation of the brain is impaired. With these conditions there is pain in the head, dreams, delirium, maybe convulsions, and a tendency to pass into coma. In such cases, when we examine the brain after death, we find anæmia, venous congestion, and, with the microscope, evidence of serous and corpuscular exudation.

We are never surprised if the patient goes right off his head—becomes insane from failing heart. Dr. Savage has told me that many patients with heart disease spend their last weeks in Bethlem Asylum. Melancholia is commoner with mitral affection. I take it that this means a more slow venous failure through the brain.

The lips are blue and distended, the cheeks bluish-red and full, and the skin has a yellowish aspect—there is a slight degree of jaundice. Here I may mention that there is an appearance of the skin which leads us to diagnose heart disease. It is commonly seen in young subjects who are suffering from rheumatic heart disease. The skin has a lemon-yellow tint, quite different from the saffron colour of obstructive jaundice; whilst the lips are bluish-purple, and the vessels of the cheeks are distended. "They look so well," say their friends; but their appearance leads us to think of a failing venous circulation.

When heart failure becomes extreme, albumen is commonly found in the urine—in some cases a large quantity—but in others, apparently similar cases, no albumen may be found. Students have often asked me, in the post-mortem room, pointing to a kidney with extreme venous congestion, "Why was there no albumen in the urine in this case?" Perhaps in another case, with less visible change in the kidney, "Why was there a third or half of albumen in the urine?" I do not know the cause of these differences; no clear explanation has ever been offered.

The expectoration often becomes charged with blood, but they never die of hæmoptysis. I have never seen any harm result from it except fright. It will do the patient good; it relieves congestion, and is a favourable sign rather than otherwise. Pulmonary apoplexy and coughing up of blood in heart disease have lost their terrors for me. In the only case in which I have ever known a patient with valvular disease of the heart die of hæmoptysis, it was revealed on post-mortem examination that there was also pulmonary phthisis.

What do we mostly notice in the pulse in heart failure? It becomes softer and softer. In mitral disease the pulse is small, soft, and usually markedly irregular and arhythmical. It is small in aortic obstruction; in aortic regurgitation it has the character known as "water-hammer."

If we examine the heart, what guide have we as to the failure? There is dulness to the right of the sternum, increasing and increasing, due to the distension of the right side of the heart. As further evidence that the right ventricle is excessively distended, we feel it struggling and heaving between the left margin of the sternum and the nipple, and we also feel it heaving in the pit of the stomach. There may be a pulsating liver. That is in many cases due to regurgitation from the right ventricle into the right auricle, and down the vena cava into the hepatic veins; but it may be due to transmission of pulsation through the valves.

Another symptom we notice, as the skin venules become overcharged, is that sweating comes on. Some of the most extreme sweating met with occurs in such cases, and the patient shrinks away. Extreme sweating should always lead us to examine the heart.

In advanced states of heart failure, we are always on the watch for acute inflammatory changes, which are very liable to come on in the venously congested organs. It may be acute nephritis, or acute pneumonia, acute inflammation of the skin, often of the scrotum, or acute gastritis. If there is acute peritonitis we are not surprised, but acute pleurisy is much more common, and in rheumatic cases acute pericarditis.

Last of all, there is more and more brain failure, there is extreme sweating, the pulse becomes almost imperceptible; it runs and runs, so that we cannot distinguish it beat by beat. For centuries that running pulse has been observed and commented on by medical men, and from it we always learn that there is a most extreme degree of heart failure, and that the fatal termination is near at hand.

I must say a few words about angina pectoris, especially as the account given in books is not very clear.

There are two groups of cases. In the first there is valvular or other structural disease of the heart, gross and readily appreciable structural disease. There is either aortic obstruction and regurgitation, with more or less dilatation and hypertrophy of the left ventricle, or it may be dilatation and hypertrophy of the left ventricle, without any aortic vavular disease. I am obliged to be very particular here, for I am speaking about a fatal affection which kills by loss of ease.

In the second class of cases there is during life no apparent sign of structural decay in the heart, no enlargement, no bruit, no sign to warrant us in saying that there is, to use the technical term, organic disease of the heart. Nevertheless, the patient dies by failure of the circulation.

At the post-mortem examination in cases of the first kind we find the usual morbid appearances of aortic valvular disease, atheromatous and calcareous changes. I have never met with angina pectoris in connection with rheumatic changes in the valves. If there is no valvular disease, we find the usual appearances of dilatation and hypertrophy of the left ventricle, with or without granular kidney.

In the second class of cases there is little or no sign of morbid change in the heart—no valvular disease, and no dilatation and hypertrophy. When minute examination of the heart was made in such cases, in some of them the coronary arteries were found atheromatous and calcareous. Then for some time it was thought that angina pectoris, when not due to aortic valvular disease, nor to dilatation of the heart, was due to disease of the coronary arteries. But I have often pointed out to you in the post-mortem room coronary arteries like rigid tubes, and on inquiry we found that there had been no symptoms of angina pectoris during life. Moreover, in the bodies of other patients who died from angina pectoris, no such changes could be found, and so that theory had to be given up. Finally, this conclusion has been arrived at, that not in gross structural change, but in the nervous working of the heart, lies the cause of angina pectoris. It has been said that the ganglia of the heart are diseased, but this is purely hypothetical, and it is better to put it as I did just now, that a change in the nervous working of the heart is the cause of angina pectoris. So the matter must be left for the present.

To understand angina pectoris we must follow the actual suffering. A patient may be walking about, and commonly is feeling much as usual; he is, however, doing something which is putting a little tension on his breathing, perhaps going up a slight ascent; he is rapidly seized with tightness across the chest, which quickly culminates in pain. Yet when you talk to the patient about what he felt during the attack, he will tell you that it is not altogether what he would speak of as pain—it is not like sciatica, it is not like toothache, it is not like being cut with a knife. He has a dreadful feeling that the chest is immovable, he feels as if it were locked, so that he cannot draw a deep breath. The agony increases and increases, and he dreads to move. Once a patient coming to see me at Victoria Park Hospital was seized with an attack on the road. When he reached the hospital and told me, I asked him what he did when the attack came on. He said, "The only thing I could do was to get hold of the railing, the nearest thing to me, and cling to it as firmly as possible. I couldn't move." If asked, "What position does a patient seized with an attack of angina pectoris when lying in bed assume?" say, "He sits up, leaning slightly forwards, with his arms down and fixed to get a position of certainty." He dares not move.

During the attack there is evidence of rapid failure of the circulation. At first the pulse is unduly full and the tension increased—it is a full, bounding pulse, but soon it begins to get very soft. There is great distress in the face, and the body is bathed in perspiration. You will probably never realise what profuse sweating is until you have seen an attack of angina pectoris. After it is over you can wring out the patient's shirt as if it had been dipped in water. Vomiting may occur. Such an attack may last for a very little while, terminating rapidly in death. In other cases wind is brought up from the stomach, the patient becomes able to move, and gets through the attack.

It is well-known that the patient may die in the first seizure of angina pectoris. In other cases he may recover from the first, but die in a few weeks, or months, in a second or third attack. If the patient lives through several attacks the case often lasts for years.

In connection with atheromatous disease of the aortic valves we sometimes see suffering which throws some light on angina pectoris. After a patient has suffered from disease of the aortic valves perhaps for a long time, a time comes when unbearableness is a marked feature of the disease day by day, cutting life short. The patient throws his arms about, moves his head about, groans a great deal, and cannot sleep. He gets weaker and weaker, and usually dies very quickly. The condition is one of unbearableness, not of pain; pay attention to that fact. I have never known that condition in connection with mitral disease, but have only met with it in cases of atheroma of the aortic valves. In mitral cases there may be extremely severe silent distress; the heart is ceaselessly knocking against the ribs, night and day they have to sit and bear it, and they get so melancholy that they can hardly speak.

Many a patient who goes to the doctor thinking he has heart disease has not what in technical language is called organic heart disease, but there is some hindrance to the easy working of the heart. Often there is some disturbance in the regularity of the rhythm; a beat is dropped now and then, or else one or two beats are more hurried than the others occasionally. And hurry is at the bottom of the mischief—the patient is hurried and harassed in his daily doings. One young man who came to me with such a condition was an athlete, and in his hurry and his eagerness he had driven his heart-rhythm. But altered rhythm is not necessarily irregular, but may be excessively stiff, a tight irritable knocking against the chest, accompanied by a tight irritable feeling in the head. If this goes on the heart may fail a little in power, or become excessively irregular. These conditions are technically termed functional disorder of the heart. With such functional disturbance the patients commonly complain of nervousness. What do they mean by that?

I was reading the *Times* one day—reading an article on some great man—I forget who it was—and the writer of the article said: "It has been contended that all great men are nervous, that the world's business is done by nervous men." This is true, for the "nervous" is that which makes things correspond with one another, and what we know as greatness largely depends on this fine power of correspondence being keenly developed. But where there is the highest functional activity there is the greatest liability to disease, and hence with the abuse of the nervous, great suffering arises. I shall consider this subject more fully when I come to lecture on nervous disease.

What the patients mean when they tell you that they are nervous is that their easy working is interfered with. We must remember that there is both use and abuse of words, for it requires careful listening to understand what they mean. At first, perhaps, they use the word in its conventional sense, and then, later, they use it in its natural sense. If you do not listen carefully and understand how to grasp the meaning of words, you may fall into the mistake of thinking, "Oh, it's of no use listening to what patients say, they tell such lies!"

We come to this conclusion, that, in order that a heart may work easy, rhythmically freely moving, there must be a healthy nervous correspondence with the world without. Now, in angina pectoris, when we follow the facts, we learn that it is the outcome of great mental suffering, usually of great worry and anxiety. It does not occur in young persons, who are mostly free from such troubles. From this we get increasing evidence that the heart may be killed, may cease to act, because this healthy nervous correspondence is impossible.

What are the facts about objective failure of the heart? After death by shock, as in compound fracture of a limb, fractured pelvis, burns, or other severe injury; after death from irritation of a serous membrane, as in pleurisy or peritonitis—in all these cases the rule is to find that the circulation has come to a stop because there is a kind of tetanic contraction in the left ventricle of the heart; the blood has been cut off from it, and we find it firmly contracted and empty. Therefore, in all cases of injury, you must never be guided by the local damage in estimating the severity of the injury, but you must ask, How much has this injury interfered with the normal tension of the body, and with the working of the heart?

The circulation stopped because the left ventricle could not pass into diastole, could not draw the blood through the lungs. The reason doubtless is that the blood could not get sufficiently into the coronary arteries to dilate the ventricle. When I was in the stage in which you are now, gentlemen, I used to think of diastole merely as a relaxation, that the ventricle simply ceased to contract, and got flabby, and was dilated by the blood forced into it by the left auricle. That is what we were all taught. Then an Italian physiologist, whose name I forget, began to throw doubt on the passive nature of diastole. We now consider that diastole is an active process, that the ventricle is opened out again as the coronary arteries are filled from the aorta. This much is certain, that as the blood is cut off from the right side of the heart, diastole ceases to be possible. I have seen cases in which the ventricle had made the attempt to pass into diastole, but had stopped halfway.

Why is there this failure of the heart? The air-passages are open. In these cases of injury we hear the air passing freely into the lungs. But what is the temperature? Perhaps it is only 94° or 95°, and lividity and extreme breathlessness come on, but there is no disturbance of consciousness—the mind remains singularly clear for a while. This is known as death by collapse. We see the same phenomena in perforation of the stomach; there is plenty of air going into the lung, but we know that there is great loss of heat by the low temperature. What causes death? The water (blood is mostly water) cannot travel through the lungs into the left side of the heart. This failure of circulation is due to loss of heat—of this there is clear demonstration.

If we are in a train, and the engine-driver lets the fire get too low, the water cannot circulate properly in the boiler, and the train slows down. Then he shovels on coals, and we see a lot of smoke come out of the chimney, and the heat gets to the water, and the train goes faster.

Why do so many infants die in cold weather? Why does a bat slink away into a hole in a tree in winter? A sunshiny day comes, and he comes out, thinking it is summer, and then he cannot get back to his hole, because he is overtaken by cold. We see the same thing when we study snakes, chameleons, bats, and human beings: they are all alike dependent on the sun's heat. Therefore in all cases of shock go for heat, and not for heat only, but for electricity.

In such cases we can see the value of a doctor. Sometimes by a word we can give the needed help; we can bring a manly spirit. Never overlook your personal influence in such cases. It may be that the kindest thing you can do will be to sit by the patient and say nothing, and he will have confidence in you, and after half an hour he may say to you, "Now I'm much better, doctor." Give them confident restfulness until the shock is counteracted.

In speaking of diphtheria and of bronchitis, I told you that as the air-passages are filled up by exudation, the blood is cut off from the left side of the heart, so that at the post-mortem examination we find the left ventricle firmly contracted and empty. The same things occurs when the trachea is blocked by a foreign body. It is evidently indispensable that the air should pass freely in and out of the lungs for the heart to be able to go on working—it tends to get spasmodically contracted as the air-supply diminishes. Therefore let the patient have plenty of air when failure of the heart is threatening.

I remember one day Mr. Sequeira called me in to see a case of emphysema and bronchitis. The patient was desperately ill. She was livid, the pulse was very weak, she was propped up in bed, and it was evident that the circulation was failing. He said, "What are we to do?"

"Senega!" I thought, "what's the use of senega? Besides, she's sure to have had bucketfuls already! If I could just take off all her things and dash cold water over her, she'd gasp and get air into her lungs, but they wouldn't understand it if I stripped her naked!" So I told them to sponge her skin with hot water freely, and after a time she began to expectorate, and she rallied.

This use of hot water was in a way imitating summer. Then if you can only make things a little more bright—put a little spring in with your hot water—a little hope—a little fresh colouring in the feeling! Therefore I say to you again, "Don't underrate the influence of your own personality." Learn to give confidence to your patients. On his way to become a medical man the student has to pass through three stages. First he doesn't know; then he thinks he knows; then he knows he doesn't know, but he stands on his feet like a man, and gives confidence to his patients.

We will return to consider failure of circulation. In phthisis death is always due to the inability of the left ventricle to go on contracting. Phthisical patients do not die of suffocation, plenty of air can get into the tubes—there may be most extensive lung-destruction, and yet the patient live for years. In phthisis we seem to have a demonstration that the plasma of the lung must remain in a fairly healthy condition to enable the air and water to commingle, for in phthisis as the plasma of the lung becomes diseased, the blood loses its power. When the blood has so lost its power that the ill-fed heart is unable to keep up the circulation, the patient dies. That is not death from suffocation. We see the same thing in anæmia—the blood loses its power and the pulse becomes softer and softer, and then the fatty disintegration of the heart comes on. The basis of giving iron in conditions of heart failure is to promote oxidation—to keep up the power of the blood.

What brings about the fatal failure of the heart in mitra valvular disease? I have told you that we find the wall of the heart exceedingly leathery, especially the wall of the right ventricle. It is charged with granular matter because the blood could not get through the coronary veins. On examining the muscle with the microscope we can hardly see the striæ. In cases in which the heart failure has been more rapid we find only œdema of the wall, and if the failure has been exceedingly rapid we find simply great distension of the coronary veins, with hæmorrhages.

We infer that to keep the heart working well, and to keep it from fatal failure, there must be a free flow of blood through the coronary veins. They cannot get their blood on because the right auricle is choked with blood—they swell up just as do the visible veins in the neck; the right auricle is choked because the right ventricle cannot get the blood on through the lungs. We see that we have always to consider the action of the right ventricle.

The most fatal hindrance to the action of the right ventricle, a hindrance which may alone kill it, is Fear. This is where anxiety tells, for anxiety is simply fear. There is something that the human mind cannot take trustfully and easily, and by degrees the breathing gets more impeded.

The point we have to consider is this. How is it that patients go on for years with valvular disease, and then get rapidly worse and die in a few weeks? Perhaps there is endocarditis or some other acute complication, but what practically kills? We very often find that the patient has been indoors too much—that he has been worried and overtasked in his nervous working—that he has been getting anæmic, with deepening depression. How are we to help him? You will sometimes be much encouraged to find how much relief the patient will get from a little aperient medicine, or from bringing up some wind. Never overlook tension in the belly. Relieve tension in the belly and you will relieve the rhythm of the heart. Or you may relieve the water tension by giving a hot bath and sweating, and this may give the patient much relief.

This gives us some clue as to when to use opium and when to use alcohol. When you want to keep the tension working easy, and the belly tension has been relieved, and the skin tension has been relieved, still you have got to help the feeling to bear it. In spite of all the new remedies for angina pectoris—nitrite of amyl and the rest—nothing has held its ground in the treatment of these paroxysms like a glass of hot spirit and water.

Alcohol has this advantage over opium, that it is breathed out much more quickly, so that we can keep on giving it frequently. Opium stays too long in the brain and other tissues. You will remember the medical man of whom I told you before. In giving opium in heart disease the greatest judgment is required. Its use will often give the patient great relief, but give it for as short a time as possible.

In my first lecture I told you how involution follows on the old track of evolution, and in heart disease we get a striking illustration. In the process of evolution blood is formed and circulates before the heart is completely organised. In the processes of involution the heart's organization may be much deteriorated, and yet it may continue to act. It is indeed questionable if the heart can become diseased and fail altogether so long as the blood is rhythmically healthy.

It is not simply the heart's contractility which circulates the blood—that contractility is a quality borrowed from the blood—the heart and the blood are mutually dependent. Moreover, the energy of the blood which gives contractility to the heart and enables the blood to circulate must depend much on the revolving orderly actions of the red corpuscles—rhythmical, no doubt, because orderly—governed by the oxidising power of the air and by the colour power of the light. In the post-mortem room I take every now and then some blood—purple and venous blood—and expose it before the students to the air and light; in a few minutes it becomes an exquisite scarlet. From these considerations we may learn the importance of fresh air and of sunlight when we want to gain a stronger circulation.

Further, food is an immense means to promote the function and movements of red corpuscles. In food, fresh from the earth, are exquisite influences of heaven and earth for the making of human nature—to encourage human feeling, and to feed the blood and heart.

Again, it is traceable that the colour of the blood and restfulness are related; that comes home to our minds as we study anæmia and mental diseases. Rest is the rhythmical government in the body—certainty in the feeling—it is easy, comforting, and refreshing, because certain, natural and orderly. All things in Nature get colour by rest.

When I knew less, in fact, nothing, about it, I thought of rest as "doing nothing." It is really "not hindering anything"—it is perfect rhythmical progress.

The so-termed physical conditions of the body are so true—true as steel—that they rest if "the mind" will let them. Often I have made that assertion to patients, to ascertain if I was thinking correctly, and the response I received from them gave increasing strength to my conclusions. And the whole art of a medical man consists in leading persons to rest.

This Buckle, in his "History of Civilization," expresses excellently, when he lays it down that the pathologist must teach how suffering comes about; but the medical man must have the tact himself and the touch of the patient to lead to health; then his own restfulness guides the patient to rest.

LECTURE LI.

KIDNEY DISEASE.

In studying kidney disease we have to remember that the great office of the kidney is to regulate—to make work safely—the water tension of the body, and that in this action it co-operates with the lungs and skin. Both the bowel and the stomach may, under circumstances of extreme disorder, be brought into corresponding action as auxiliaries. The degree of activity of each of these tension-regulators will depend on the varying needs of the general circulation. In hot weather the water-function of the skin is exceedingly active, and perspiration is profuse, the water-function of the lung is also very active, and little water is passed by the kidney. In cold weather, on the other hand, the skin and lungs are less active, and much water is passed by the kidney. Similarly in disease, if there is much watery evacuation by the bowel or stomach, the urine may be much diminished. This is very impressively witnessed in cases of strangulated hernia. When there has been vomiting for days, suppression of urine may come on; but when the strangulation has been relieved, and the vomiting has ceased, and the patient is able to drink water, very soon urine is again passed abundantly. Again, in cholera, after the patients have passed a large quantity of water by the bowel, suppression of urine commonly comes on; but as recovery from collapse occurs, urine is again freely passed.

In collapse from any cause, as in perforation of the bowel, or after severe injury, as the general circulation fails, and the flow of blood through the kidney is arrested, the secretion of urine ceases. In conditions of failing heart, also, the secretion of urine diminishes, and if the heart gets stronger the secretion is re-established.

The secretive or excretive function of the kidney (we can regard the kidney-function from either of these points of view—it removes from the blood effete material and passes onwards purified blood) is dependent on respiration, which enables the blood to circulate through the kidney, and promotes the functional activity of this organ. Hence, when persons are confined in a close atmosphere, or when, owing to the condition of the lungs, as in vesicular emphysema, the air-supply to the blood is diminished, the function of the kidney is, as shown by the urine, disordered. It is demonstrated in Nature at large, that air moving through water immediately lifts it; in other words, the travelling air circulates the water, the air itself being moved by heat-energy. It is, then, presumable that diminished air-supply, not only by diminished oxidation, but also by diminished diffusion, tends to lessen kidney function—the venous circulation being weakened owing to the gases not passing sufficiently through the water (serum) of the blood. Further, one indispensable aid to healthy respiration is the easy working of vis nervosa; and hence, if nervous energy be much disturbed, kidney function is commonly disordered. In some such cases there is a diuresis; in other cases, with nervous disturbance the urine becomes very scanty, as may be seen in some cases of hysteria and insanity. With insane conditions I have known suppression of urine without any sign of nephritis. I remember a case in which there was extreme mental disorder, and there had been pneumonia and rheumatic gouty changes in the joints. But the urine was repeatedly examined, and was found free from albumen; the colour and specific gravity were normal, and antecedent to the suppression the urine was passed in normal quantity. Suppression came on a week or so before death, and I was led to connect it with cerebro-spinal failure.

It is also found that food, drugs, and notoriously alcoholic compounds, very much affect the kidney functions. The degree of activity of the muscular system also reacts on the kidney function, directly by influencing the venous circulation, and indirectly by increasing the excretory products.

We may discern that disorders in these several conditions tend to bring about disorder of the kidney, until at last there is "organic disease"—a decayed, worn-out kidney.

We will now go on to consider *suppression of urine*, which usually comes on as a consequence of organic disease of the kidney. It may either be a chronic tissue decay, as granular contraction of the kidney, or it may be acute inflammation, as in large white or mottled kidneys. As I have said, it also comes on in some mysterious conditions in which there is great disturbance in the nervous system, and evidence of spinal cord failure. In such a case, while making the post-mortem examination, I have heard the house-physician say, "This patient died from suppression of urine," and I have been surprised to find how little morbid appearance there was in the kidneys.

The first thing noticed in suppression of urine is that the secretion becomes more and more scanty, until only a few ounces, or perhaps none at all, are passed daily. As the quantity of the water diminishes, its specific gravity rises—the urine becomes condensed, and it may be so much so, that a catheter has to be passed into the urethra to clear away earthy matter which has collected. It has astonished me in cases of suppression to see how little the patient seems distressed—in no pain, talking as usual, not looking extremely ill. And I have experienced difficulty in leading the friends to believe that the patient was in imminent danger. There was little or no vomiting, very little complaint of shortness of breath, but increasing weakness and failure of the circulation; no loss of consciousness until a few hours or minutes before death, and no delirium until just before death. It is surprising to find the mind so remarkably calm.

For many years we have been taught that the coma of so-called uræmia was due to failure of the excretory function of the kidney. But in these cases of suppression there is extreme failure of the kidney without coma. Sir William Gull and I drew attention to that fact at the Medical Congress in London.

In cases of suppression I have been most impressed with the signs of increasing failure of general circulation, and of failure of muscular and nervous energy; in other words, by the rapid loss of strength. In some cases, beyond question there had been inflammatory changes in the kidney to account for the suppression, and consequent failure of general circulation. In others, however, in what degree the suppression was due to antecedent failure in the general circulatory energy, or in what degree the failure of circulation was due to the suppression, I could not see my way to answer.

The kidneys, like the lungs and other organs, are advantageously studied according to their tissues. They have a mucous surface communicating directly with the bladder and the outer world; they have a sero-vascular surface communicating with the great sero-vascular channels and structures of the body; between these lies the fibrous layer communicating with the connective tissues of the body at large; of the nerve tissues I cannot speak. The causal conditions of kidney disease are grouped according to these tissues, and the symptoms vary according as the disease affects mainly one or other surface. The great office of the mucous surface of the kidney is to transmit water, holding in suspension the effete, useless products of tissue activity, and getting them out of the body; when the mucous surface fails in this function the consequences to other bodily functions have now been very clearly ascertained.

Let us first consider the so-termed *surgical kidney*, suppurative pyelitis and nephritis. It is a very common condition. The suppuration usually begins in the bladder, extends up the ureter, and attacks the pelvis of the kidney. The pelvis of the kidney becomes thickened by inflammatory exudation, pigmented by extravasated blood, and softened until disintegration occurs. The suppurative inflammation travels along the cones to the cortex of the kidney, and collections of pus are found in the cortex. This process may have gone so far that the kidney substance has been almost entirely broken down into abscesses.

It is necessary to bear in mind that these suppurative processes, which usually commence in small areas, are commonly repeated perhaps for months; and that, with each fresh outbreak of suppurative inflammation there is rapid increase of tension in the kidney. We are guided to think that this is so as we peel off the capsule of the kidney, and observe how tightly packed, evidently stretched, the kidney substance is within the capsule. During life we are guided to the same opinion by the fact that in many cases the patient is attacked with repeated rigors—every few days there is an attack of shivering, followed by heat and sweating, as in ague. At the period of the rigor we notice increase or recurrence of the pain in the back, suggesting increased tension in the kidney, and at such times the urine is increasingly charged with pus.

As such symptoms are repeated, we notice that the skin of the patient becomes yellower, he is more cachectic-looking, and he feels weaker, but retains clearness of consciousness. There is commonly no coma, and there is no delirium, as in uræmia. Weakness is the most prominent symptom. There is no dropsy, there is little or no vomiting, shortness of breath is little or not at all complained of. In all these respects it is strikingly different from Bright's disease. The tongue becomes red, owing to catarrhal inflammation, and there may be diarrhœa, the skin becomes drier, the breath smells of urine—ammoniacal. This morbid condition has been by some called ammonæmia—blood-poisoning by decomposing urine. These patients die of asthenia. Numbers of times I have listened to the house-surgeons giving such a description of the symptoms which had occurred in fatal cases of stricture and other diseases of the urinary passages. We most commonly meet with such cases in connection with stricture, but they occur in cases of spinal cord disease, where there is paralysis and suppuration of the bladder, and in cases of carcinoma involving the bladder, also in fibroid tumour of the uterus, or ovarian, or other tumour pressing upon the bladder or ureter.

In these cases the difficulty is to keep the blood from being poisoned. The pus rapidly decomposes and noxiously infects the system. Therefore do not overlook that such kidneys want much water and that we should supply them with this to wash away the pus and decomposing residue, and that the patient requires rest to enable the renal circulation to travel easy enough to pass out sufficient water. For that end the kidneys must have sufficient blood, at the required tension, passing through them.

Before leaving this morbid condition, let me repeat that, unlike uræmia—so-termed—although the body is thus poisoned, yet the patients are not comatose nor convulsed, and they die from muscular and nervous failure. Remember that suppurative pyelitis may be secondary to stone in the kidney, and that in some such cases a large abscess may be formed, which may either open through the skin, or externally. Suppurative nephritis may also be due to strumous change.

This leads us to consider *strumous disease of the kidney.* It may take the form of simple catarrhal inflammation, albuminuria with malaise, lasting two or three weeks, and passing away like an ordinary cold. Secondly, it may take a more insidious form, and pass into a condition known as large white kidney. Coming on insidiously, the patient may be walking about with much albumen in the urine, but getting paler and paler, increasingly anæmic; after weeks, dropsy sets in, and we say that it is large white kidney occurring in a strumous subject, because the patient suffers from strumous disease of lung, bone, or other organs.

The third typical form of strumous affection of the kidney begins in the pelvis of the kidney, and hence the disease is generally known as strumous pyelitis. The changes are very similar to those which occur in strumous disease of the throat and other parts. The mucous membrane becomes swelled and charged with granular exudation. There is venous congestion, and the distended venules give the membrane a blueish appearance. As the swelling takes place the epithelium becomes detached, thus giving the membrane a granular appearance, and the denuded surface becomes covered with pus. Whenever we find such a condition of the pelvis of the kidney we immediately look for something to account for it. We find no evidence of suppurative inflammation extending up from the bladder, and we think it is strumous. On further examination of the kidney we find caseous matter, the so-called yellow tubercle, in its substance. When we examine the lungs we very commonly find phthisis; often also there is strumous ulceration of the ileum. The morbid change also very commonly extends from the kidneys into the ureter, and it may reach the bladder and even the urethra. There is then caseous matter and ulceration throughout the urinary passages.

As the morbid change in the kidney increases, the kidney is swelled with more and more granular deposit; then acute inflammation arises around this strumous material, and it breaks down into pus. The appearance of such a strumous kidney is very characteristic. The cortex and the cones are charged with and swelled by a yellowish-grey granular material. In this there is scattered caseous material, and the kidney substance is riddled with abscesses. In this way a kidney may be entirely destroyed. Nothing may remain but the fibrous capsule and the fibrous remains of the pelvis. Strumous inflammation may affect one kidney only, but it commonly attacks both. Strumous inflammation may attack the bladder primarily, and in such a case may spread up to the kidney.

We have to recognise also that strumous changes in the kidney, like strumous changes in the lungs and in other organs, are commonly arrested, and the patient may recover. I have found remains of old strumous disease of the kidney when the patient has died of some entirely different condition, and from the clinical history of the affection we also learn that it is a curable condition.

This brings us to consider the actual suffering in strumous disease of the urinary organs. Where the bladder and urethra are affected—and I have told you that this may either precede or follow the kidney affection—there is commonly pain in passing water, and discharge of pus. Be sure and not jump to the conclusion that gonorrhœa is present because pus is so passed. Smarting when passing water is one of the common symptoms in kidney disease. The strumous inflammation of the bladder give rise to frequent desire to pass water, and other symptoms of bladder disturbance.

As the kidney substance becomes increasingly swelled, pain is a prominent symptom, pain in the back; the temperature is raised; there is discharge of pus in the urine day after day, week after week. If the inflammation be very acute the patient may pass much blood; if, therefore, a patient come to you with protracted hæmaturia, with great wasting, and pain in the loins, immediately think of strumous disease of the kidney. In some cases we may be afraid that the patient is going to bleed to death; one of the most severe and protracted forms of hæmaturia next to that met with in cancer, is due to strumous kidney. As the patient goes on passing quantities of pus, and the urine is charged with it, we say it is not Bright's disease, and as a rule dropsy does not occur; in fact, dropsy never occurs with acute suppurative nephritis—at least, I have never seen dropsy where a large quantity of pus has been passed week by week. Just at the last there may be œdema of the lung, but this is common to all forms of slow death. The other symptoms are merely those of asthenia, and in some cases we may be able to feel the kidney tumour. The variable nature of the pain in such cases evidently depends on the variations of inflammatory tension. These diseases may recur for years, the kidney healing and becoming diseased again; or the patient may die in a short time with acute strumous disorganisation of the kidney.

Lardaceous disease of the kidneys may always be recognised by its association with lardaceous disease of other organs, especially the liver and spleen. The kidneys are large—usually very large—and directly we see the surface we are struck by a peculiar purple-red, blotchy appearance, mixed with a shiny yellow substance. The cortex is waxy and thickened. On adding iodine we find that the region of the cones stains most deeply. With the naked eye, however, the result of the iodine reaction in lardaceous disease of the kidney is disappointing, but under the microscope a section of waxy kidney treated with iodine has a most striking appearance. A characteristic appearance under the microscope is that the whole kidney seems infiltrated with a hazy granular material, amid which the Malpighian bodies stand prominently out.

In lardaceous disease of the kidneys we do not meet with as much œdema as in Bright's disease, and death in lardaceous disease usually occurs from failure in the mucous membranes; in these lardaceous change occurs, and catarrhal inflammation frequently arises, the absorption of food is arrested, and death ensues from mal-nutrition and exhaustion. Ascites is common, and, at the last, œdema of the lung.

I wish I could tell you more about the way in which a kidney works. If we knew more of this, we should be in a better position to understand the changes which bring about disease. All we can say is, that a healthy kidney works in such a way that we do not know that it is working, and that there are many gradations between a healthily working and an organically diseased kidney.

A man of about middle age goes to an insurance office. He thinks he is quite a healthy man, but when his urine is examined it is found to contain albumen, and the life is refused. He went into the place thinking he was healthy, and now he cannot insure his life at any price, and he thinks he must be very ill. Years later however he finds he is not any worse, and he thinks of getting married. He marries, and after a time he meets the doctor whom he saw at the insurance office, and he damns him well, saying, "Why did you want to make me go for so many years in fear of my life."

In the American insurance offices it has been astonishing to find the large number of apparently healthy people who have albuminuria. Our resident medical officers often get it, and get rid of it. Hence we are led to think that one of the common functional disturbances of the kidney leads to the appearance of albumen in the urine. We learn that, in addition to the disintegration in the kidney, producing what is known as organic disease, there are temporary disturbances in the performance of the kidney, which we call functional disease. What are the causes of such disturbance?

It may be, and very commonly is, secondary to nervous disturbance, such as exhaustion of the spinal cord and brain. which a few day's rest and change of air will remove. Functional albuminuria may be brought on simply by the agitation which the patient suffers in going to see the doctor. I was once spending an evening with Sir William Gull, and he showed me a specimen of urine, saying, "Look at this when I boil it." It contained a large quantity of albumen, and he said, "Now, is that due to functional or to organic disease?" "I don't know," said I. "That urine," said he, "loaded with albumen, is passed in the morning. The urine the patient passes at noon contains very little, and that passed in the evening is free from albumen."

"Then," I said, "I have no doubt that it is functional albuminuria, and I should not attach much importance to it." He told me that, for other reasons, he had come to the conclusion that there was nothing more than functional disturbance. Such morning albuminuria is not uncommon. Where there is nervous excitement it is always greater in the morning, and becomes easier as the day goes on.

We are never surprised to find transient albuminuria in cerebro-spinal disease. Albuminuria may also be due to weakness of the venous circulation, and also to poisoned conditions of the blood, such as malaria and syphilis. Albuminuria may come on during the persistence, or after the disappearance, of a chancre. It is very transient, leaving no sign of nephritis, and seems therefore to be due to a morbid condition of the blood. Albuminuria occurs in connection with gout. If it only appears during the attacks of acute gout, and in the intervals the urine is free from albumen, we cannot say that there is any permanent disease of the kidney. Albuminuria also occurs in connection with abuse of alcohol, in cases in which there is not as yet any organic disease. We may find albumen in the urine after a long walk, or a hard reading-bout.

How is it that albuminuria is not more common? This is a question which physiologists have often asked themselves. We now know that it is much more common than we used at one time to think.

We also meet with an extreme excretion of lithates denoting functional disturbance of the kidney. Sometimes this is accompanied by albuminuria, sometimes not. It is common in connection with nervous disease. I have heard a patient say, "I know I've got one of my headaches coming on." "How do you know that?" I asked. "Because my urine was thick this morning with a brick-dust deposit. It's always like that before one of these attacks." In some cases of functional disorder we meet with an excessive excretion, not of lithates, but of phosphates.

In what other conditions do we meet with an excessive excretion of lithates? In cerebro-spinal disease; in venous obstruction, as in heart disease, in vesicular emphysema, in pressure on the inferior vena cava, and in extreme ascites, in which the renal veins become compressed by the effusion; when the blood is rendered very unhealthy, as for instance, in liver disease — we meet with a typical brick-dust deposit in cases of abscess of the liver; in fevers, brought about in two ways, by venous congestion, and by diminished oxidation; where the skin function is much irritated, as in very hot weather.

Sugar is found in the urine under many different conditions, and up to the present time the tendency has been, in many instances, to over-estimate its importance. Does glycosuria always mean diabetes? Certainly not. When does it mean diabetes? Only when the other characteristic changes of diabetes are present—1. Much wasting. 2. Much disturbance in the nervous system. 3. Much catarrh of mucous membranes. 4. Drifting on to organic disease of the lung.

Glycosuria may be due to very temporary causes, to nervous disturbances. We are never surprised to meet with it in cerebro-spinal disease. It occurs also with liver disease, especially cirrhosis. It occurs with lung disease, especially phthisis, and it is often very puzzling to distinguish such cases from true diabetes. The conditions leading to such functional glycosuria are as yet undetermined.

What are the characters of the urine where the kidneys are venously congested? 1. It is scanty, because the blood cannot easily get through the kidney. We are sure that the blood must flow freely through the kidney, in order that a proper secretion of urine may be formed. 2. It is loaded with lithates —a condensed urine, of high specific gravity. 3. It commonly contains albumen, but the quantity of this is very variable. 4. Hæmaturia is rare, though with the microscope we find hæmorrhages into the tube where there has been prolonged venous congestion of the kidney; but when I speak of hæmaturia, I mean a condition you can detect with the naked eye. If there is hæmaturia evident to the naked eye, we always suspect that nephritis has come on.

We will now consider *Bright's disease*, and I think that, perhaps I shall convey to you the clearest notion by recounting briefly a few historical particulars of the study of the relations of albuminuria.

It had long been recognised that when patients became swelled by dropsy, there was something wrong in connection with the passage of their water. Later it was recognised that such patients passed in their water something which, when boiled, looked like white of egg, and was therefore called albumen. This was the stage which clinical investigation had reached when Dr. Bright began the study of these diseases at Guy's Hospital. His investigations showed that there were destructive morbid changes in the kidneys themselves. From that time, when practitioners found albumen in the urine, they said there was "kidney disease." For years albumen in the urine was considered to be unquestionable evidence of kidney disease—and so it doubtless was, using the words in their proper meaning—but what was then meant by "kidney disease" was renal tissue decay. After a time it was ascertained that such decay was by no means present in all cases of albuminuria, which might be due merely to disordered function. The conditions under which such functional albuminuria arises have been already considered.

Bright further observed, that in some of the cases there was no dropsy, but in others there was much. This led him, and subsequently other observers, to search further, and they recognised, that in the cases in which there was much dropsy, there was usually much albumen in the urine, but in the other group of cases, in which there was no dropsy, there was usually less albumen, and at times none at all.

He further noticed that where there was much dropsy and much albumen there were usually large white kidneys. Where there was no dropsy, and a variable amount of albumen, there were usually small granular contracted kidneys. He concluded that there were two natural groups of cases. In both the symptoms might extend over months or even years. Pathologists agreed to speak of these cases collectively as chronic Bright's disease.

His observations went farther, and demonstrated that in many of those cases which had large white kidneys, there had been blood in the urine, the urine had been scanty, and the illness had come on in some cases with a febrile outburst, and evidently acutely. Such cases it was agreed to speak of as acute Bright's disease.

Further observation showed that acute nephritis very commonly—I may say, most commonly—supervenes on some chronic degeneration of the kidney. By acute Bright's disease we really mean acute nephritis, which may be secondary to chronic disease of the kidney, or may be a primary affection, arising in connection with scarlet fever, syphilis, or other acute malady.

Here I ask you to allow me to acknowledge my debt to Dr. Wilks, whose labours in this and in many other subjects have greatly advanced pathological teaching. He taught me pathology, and showed me that acute inflammatory changes are generally consequents of chronic disease; it was his mind that showed me how to study morbid changes scientifically.

As post-mortem examinations went on, another effect was noticed. There are cases in which much albumen is passed, and there is very little dropsy, but there is a large liver and a large spleen, and we usually find either bone disease or syphilitic remains or phthisis, and we call such kidney alteration, from its appearance, lardaceous disease of the kidney. Of this I have already spoken, but some observers think it more convenient to group that disease under the term Bright's disease.

Next let us inquire what conditions produce *acute nephritis.* Does exposure to cold produce acute nephritis? As far as I have been able to gather, exposure to wet and cold, where this is alleged as a cause, has been but the immediate determiner—other marked changes, injuries, or cachectic conditions—having preceded the exposure. Perhaps a patient is threatened with rheumatic fever or with phthisis, and gets acute nephritis. But we see no reason to think that healthy subjects ever get acute nephritis from cold. Most commonly there is antecedent chronic disease, either in the kidney or elsewhere in the body. Frequently acute nephritis supervenes on a condition of chronic venous congestion of the kidney, and it therefore arises in cases of valvular and other heart disease, in vesicular emphysema, in phthisis, in fibroid degeneration of the lung, and in dilatation of the bronchial tubes. In all these diseases it is important to be on the watch for acute nephritis, and the urine should be examined from time to time. Acute nephritis often comes on in association with blood-poisoning and other cachectic conditions, such as scarlet fever and diphtheria. In these it is most common, but it may arise after measles, small-pox, or any other contagious fever. In these cases venous congestion is doubtless more or less operative, in addition to the blood-poisoning. Nephritis similarly liable to come on after parturition, when the blood has a tendency to stagnate in the veins. In connection with diphtheria let me say that I have never seen nephritis where the patient died during acute diphtheria, but it often occurs two or three weeks after the onset of the disease. Syphilis is another cause; I believe if a large number of cases were carefully watched, it would be surprising to find how frequently acute nephritis arises during secondary syphilis. Such patients generally recover. Nephritis may be due to mechanical injury, such as a blow on the loins. The chronic changes in the kidney which may give rise to acute nephritis are, first, fibroid change, and hence acute nephritis is common in cases of granular contracted kidney; secondly, strumous changes, and further acute nephritis is common where there is a family tendency to phthisis; thirdly, adenoid or cancerous growths, and also chronic syphilitic changes; fourthly, embolic changes. I cannot speak definitely about acute nephritis coming on from arterial disease, excepting where the arteries are much altered by fibroid changes—in such cases, acute nephritis is very common, but there the kidney tissues have degenerated. You must never confound with acute nephritis inflammation travelling up from the bladder. This, as I have already explained, produces suppurative inflammation of the kidney, whereas there is never suppuration in acute nephritis, nor, on the other hand, does œdema accompany suppurative nephritis. In examination, I often ask this question, "Is there ever suppuration in the kidney in acute Bright's disease?" and I expect the student to say "No." Then I know that he clearly distinguishes in his mind between the two conditions, and that he will not set about washing out the bladder for acute nephritis.

In the early stage of acute nephritis the kidney is usually found much enlarged and extremely congested. I familiarly speak of it as the "chocolate-coloured kidney," because it has such a deep brown-red colour. It is extremely swelled, and very commonly there are hæmorrhagic extravasations in the cortex, so that the kidney may be studded with ecchymoses. In these cases there had been less blood in the urine, and it was evident that the vessels had become so extremely filled and stretched, that the blood had stagnated in the kidney. The clinical history also revealed that at this stage the urine had been much diminished, or entirely suppressed. Through the distended vessels serum oozes, and we therefore find albumen in the urine, and also hyaline casts, some of which have epithelial cells entangled in them—these are the so-called epithelial casts. You may readily prove the real origin of the casts by making microscopic sections, and seeing them in the tubes of the kidney.

In the next stage the redness much diminishes, and its place is taken by a grey material, an albuminous substance. Areas of congestion, intermingling with areas of this grey material, form what is known as a mottled kidney. These kidneys are usually large, and they are called "large mottled kidneys." From this we are led to infer that, as the disease continues week after week the congestion diminishes, and at last we find that the morbid change throughout the kidney is characterised, not by congestion, but by bloodlessness, pallor; and then we speak of "large white kidney."

There is a group of cases in which the kidney is most extremely pale. I have called it the "white marbly kidney." I remember the case of a girl who had been under Dr. Down's care, on and off, in the hospital for two years, and I found the kidneys in that condition, looking like white marble in colour.

It is puzzling to discern how best to speak of, and lead our minds to exact and comprehensive views of, morbid and other conditions, when they are very complicated and variously combined. The course taken must be the most convenient, the one that seems at the moment the most handy and useful. It will, I think, be best here to continue the description of the anatomical and histological morbid changes in the kidney, though time will compel me to do it briefly.

Let us first consider *venous congestion* of the kidneys, resulting from heart, lung, or other organic failure. We frequently have before us these soft bluish-purple kidneys—some of the largest kidneys we meet with. The capsule separates readily, and we find the stellate veins and the fine network of surface veins much distended. In such kidneys the venules of the cones are exceedingly noticeable from their over-distension with darkened blood, and the cones have thus a deep purple appearance. If the venous congestion has continued over weeks or months the kidneys are smaller and firmer ; they cut firmly, leaving a sharp edge. These are commonly known as " heart kidneys."

In some of these venously congested kidneys we find the cortex studded with minute blood extravasations, and this ecchymosis may be mixed with purple and grey material, leading us to inquire if, or to what extent, nephritis had occurred shortly before death. At the bedside, at the post-mortem table, and even with the aid of the microscope, it is often difficult, and may be impossible, to decide whether there has or has not been nephritis. The appearances may be quite decisive. When the vessels are over-filled, but there are no signs of excessive effusion into and swelling of the tissues, we recognise mere congestion ; when such effusions are well marked and wide spread, we acknowledge the changes to be inflammatory. Nevertheless, there are intermediate degrees of effusion and swelling. Of these, it is more or less allowable for arbitrary opinion to say there has or has not been nephritis.

With venous congestion it is very impressive to see the intertubular capillaries filled with yellow corpuscles and with scattered white corpuscles. Amid these distended vessels are imbedded the convoluted tubules, the epithelium of which is more or less clouded by granular exudation. In many other specimens, however, there are exudations of red and white corpuscles around the capillaries, and the nuclei of the epithelium are much swelled and clouded ; and in these cases we are obliged to admit that the morbid conditions had—at the time of dying, to say the least—been passing into an inflammatory outburst.

Protracted venous congestion is one of the most, if not the most, common condition leading to nephritis. Consequently where there has been serious and continued difficulty of breathing, it is common to find evidence of nephritis. We therefore often meet with it, not only where there has been valvular or other cardiac disease, but with phthisis and other diseases of the lung, with fever or diphtheria, or coming on insidiously in the last few weeks of cirrhosis of the liver or other diseases in which breathing has been laboured and difficult.

In the *large white or mottled kidney* the tubules present a swelled and clouded appearance. The epithelial cells are clouded by granular and so-called fatty matter; and the nuclei are swelled and appear to have divided and multiplied. The nuclei of the glomeruli also seem to have multiplied; and there may be such a quantity of albuminoid hyaline extravasation under the capsule of the glomerulus that it has compressed the capillaries to an extreme degree. A hyaline homogeneous material, doubtless albuminoid, is lying between the epithelial cells of the tubules, forming the so-called hyaline casts. The epithelium of the tubules may thus be so much compressed that the cells have been much destroyed; and doubtless destruction of the epithelium occurs in other ways.

You are familiar with the hyaline, epithelial and granular casts, and blood casts, found in the urine in cases of nephritis. It has been customary to consider that these casts came from the convoluted tubules, and that their presence in the urine revealed the kind of morbid change going on in the convoluted tubules. Further investigations into the minute anatomy of the kidney has, however, led to doubt being entertained as to the portion of the tubules from which the casts come, and we are therefore doubtful as to their precise significance.

With these alterations in the tubules there are indications of inter-tubular inflammation, swelling of the connective plasma, and exudations of coloured and colourless corpuscles around the capillaries and into the tubules. But in some kidneys the inter-tubular exudations, swelling, and other alterations, are much less marked, the morbid changes being mostly intra-tubular, and we are then led to infer either that in such cases the morbid changes have been much less acute, and doubtless more insidious, or that the acute changes with the exudation have passed away.

Intra-tubular changes are so commonly associated with swelling or other thickening of the capillary wall, that it seems arbitrary to divide the cases into intra-tubular and parenchymatous nephritis; and yet there is a help in so doing, for we endeavour to define where the disease began; either the morbid change was primarily intra-tubular; or it was primarily inter-tubular; so we hope to discern its further relations.

In some kidneys the inter-tubular changes are extremely marked, and denote that disintegrations have been slowly going on; the connective tissues are fibroidly thickened, the arterioles and capillary walls thickened, and the capsules of the glomeruli also thickened. Such thickening may either present a felt-like appearance, indicating that it has been of long standing, or there may be a large number of spindle cells and divided nuclei in the connective-tissue area, denoting that there has been a more recent and progressive fibro-cellular growth. In other cases these inter-tubular changes are met with in less degree. In their extreme form and extension into the cortex we recognise that these fibroid inter-tubular formations are similar to those of granular contracted kidney. Yet we have to regard them somewhat differently, for their antecedents have been different, and it is almost needless to say that effects may have much similarity of feature, yet be more or less different because of the difference in their antecedents. In some cases it is an open question as to how we are to regard the fibroid inter-tubular changes. For example, the clinical history of a case extending over years has led to the diagnosis of fibroid granular contraction of the kidneys. At the post-mortem examination large white kidneys are found, but the microscopical examination reveals fibroid changes similar to those of granular contracted kidney. The question arises—did recurring sub-acute nephritis lead to the fibroid changes, or was the fibroid the primary change, the recurring nephritis consecutive?

It is recognisable with the kidney, as with the lung and other organs, that the inter-tubular structure may be much thickened by fibroid material, and that inflammation may have recurred in the mucous tissue from time to time, as bronchitis or nephritis, but it is often questionable where the inflammation had primarily and mostly prevailed—in the mucous tissues until it extended to the vascular and connective, or the reverse.

Where strumous conditions have been the antecedents, or where granular contracted kidney has occurred in a case in which the urethra has been strictured, and the pelvis of the kidney has been dilated and the cones much destroyed, I have been led to think that the morbid change had begun in the mucous membrane and extended to the parenchyma. It is, however, obvious that the inter-tubular changes and fibroid contraction may also have been promoted by conditions, alcoholic or other, which more ordinarily produce granular contraction of the cortex.

I dwell on these particulars for the reason that intra- and inter-tubular morbid changes are so frequently associated, and because, when we have inquired as to their antecedents, we have found that they differed much. It is agreed that such changes may result from protracted venous congestion, with or without thrombic or embolic deposits. In other cases the antecedent condition has been syphilitic or scarlatinal nephritis. It would seem most probable that in cases resulting from protracted venous congestion, or from syphilitic or scarlatinal nephritis, the morbid changes would begin in both areas, mucous and inter-tubular, about the same time, and end in granular contracted kidney.

And where the appearances were merely those of large white or mottled kidneys, I have often been surprised to find extreme fibroid inter-tubular changes, the fibroid tissue looking of much older date than the tubular changes. I have been led by my experience of such cases to teach that the kidney may undergo slow fibroid changes in parts, and that whilst in that condition acute nephritis may supervene, and the kidney become extremely swelled and altered, until it looks like a large white kidney, and that in such cases the patients die with symptoms of large white kidney. In this respect the experience of the kidney is very similar to that of the lung, in which pneumonia commonly supervenes where the lung is injured by scattered fibroid change, and the whole organ then becomes swelled and otherwise altered by the inflammatory exudation,

There are many other kidneys in which fibroid changes are extremely and characteristically marked—distinctive of so-termed granular contracted kidney; and the contraction is so extensively spread throughout the cortex, whilst the epithelium of the tubules is comparatively so little altered, that we cannot but contend that the morbid changes have begun in the intertubular area. The fibroid changes are most marked in and around the walls of the arterioles and capillaries, and so Sir William Gull and I were led to regard them as beginning there and spreading thence in patches through the cortex of the kidney; subsequently the fibroid substance contracts, and by compression destroys the tubular structures. The epithelial layer here and there may be entirely destroyed, that is, some of the tubules are bared of epithelium, and their bulgings constitute the so-called cysts.

These various experiences have shown that the local organic morbid change, granular contracted kidney, is the result of various antecedents, and it is obvious that the local disease must be studied accordingly. It will be necessary for us to consider somewhat more fully the pathology of granular contracted kidney, but before doing this it will be more convenient to discuss the symptoms and complications of acute nephritis.

From what you have heard you will be prepared to learn that the symptoms of acute nephritis are commonly preceded by and associated with the symptoms of other disease. In the early stage of acute nephritis there may be a feverish outburst, but this is seldom met with. It is possible that it occurs in some cases and has passed off before we see the patient. It is certainly very exceptional for acute nephritis to come on like a "fever." Pain in the back is usual in, and characteristic of, suppurative inflammation of the kidney, but much pain is only found in exceptional cases of the nephritis of Bright's disease. In one case the medical man thought the patient was sickening for small-pox—small-pox was prevalent, and the patient had severe pain in the back and vomiting—the case turned out to be one of acute nephritis.

There are many cases in which the renal symptoms—albuminuria, hæmaturia, giddiness, weakness, and shortness of breath —have come on, but with no appreciable dropsy. Under treatment the symptoms disappear, and the patients entirely recover in a short time. It is usual to consider such nephritis as the result of cold. In other cases the hæmaturia and albuminuria are more persistent; there is more difficulty of breathing, and more or less dropsy follows, and only after many weeks do the patients lose most or all of the symptoms. In such cases it often remains an open question, how far the tissues have degenerated, and it is apprehended that the acute symptoms may recur. In other cases the symptoms are much longer persistent, they are more severe, and they have come on more insidiously. In such cases there may be indications of antecedent strumous conditions, or of much alcoholic or other abuse, or manifestations that the general circulation and tissue nutrition have been antecedently much weakened. In these cases there are the usual indications of large white kidney—much albumen persistently in the urine, much œdema, much anæmia, difficulty of breathing, weakness, giddiness, vomiting, and pains in the head. Remember then that at the beginning of acute nephritis there is usually no dropsy, unless due to heart or other organic failure, and weeks may pass over without any dropsy appearing. We then notice that anæmia, shortness of breath, and dropsy, come on at about the same time.

If you are asked, "How do patients most commonly die in acute Bright's disease?" say, "By œdema of the lungs." "In what other ways may such patients die?" "By œdema of the stomach or intestine, causing fatal vomiting or diarrhœa; by acute inflammation of various organs; by hæmorrhage; by uræmia." It will be better to defer our consideration of uræmia until we have described granular contracted kidney, but we will now speak of œdema, of inflammation, and of hæmorrhage.

Œdema comes on as the serous circulation begins to fail, and increases until the subcutaneous cellular tissue is charged with watery, serous fluid, and similar fluid accumulates in large quantities commonly in the peritoneal cavity, in the pleural cavities, and may be in the pericardial cavity, until, perhaps, there is threatening death from over-distension of these serous cavities; but most danger arises from œdema of the lung—the serum accumulating in the air-cells and tubes, and filling them more and more, until the air is shut out. This is common, not only in large white kidney, but in other forms of renal decay How do we recognise œdema of the lungs on post-mortem examination? The lungs weigh much heavier than normal; the crepitant condition of the lung is lost, and it feels semi-solid. On section, a large quantity of yellowish fluid mixed with air-bubbles oozes out, and on squeezing the lung we can drive out an enormous quantity of this. When we cut open the trachea and the bronchial tubes, we find them filled with a frothy serous fluid. Is œdema always generally diffused throughout both lungs? No, not always—it more commonly involves both lungs throughout, but it may be exceedingly localised in the apex or base of one lung.

When kidney function is failing, the lungs and skin are, for a while, able to compensate for this failure, and to prevent the body from being choked with water; but as this supplemental action becomes inefficient, œdema increases, and at last the lung itself becomes œdematous. The first symptom that this choking of the lung is occurring is difficulty of breathing, and therefore dyspnœa is a symptom for which we must always inquire in Bright's disease, and let me add that this is a sign by which we must be guided much more than by the quantity of albumen in the urine, for many cases in which a large quantity of albumen is daily passed for weeks, do well.

There is another way in which œdema may kill. The serous fluid accumulates in the mucous membrane of the stomach, and the patient is suddenly attacked with vomiting, which is repeated until collapse sets in. I remember, when cholera was prevalent, making a post-mortem examination on the body of a man supposed to have died of cholera, but there was no sign of this disease—it was a case of acute nephritis, in which vomiting had set in. Whenever vomiting in acute Bright's disease is severe and frequent, do not underrate its importance, but put the patient in bed, keep him warm, and facilitate the circulation all you can. Œdema of the intestinal mucous membrane may kill the patient by uncontrollable diarrhœa.

In inflammation there is locally a still greater failure of circulation than that which leads to œdema—leucocytes are poured out as well as serum, and also a few red corpuscles. Inflammation of the serous membranes is especially frequent, and death may occur from suppurative pleurisy, peritonitis, or pericarditis, or cellulitis of the skin may end the case fatally.

Still greater vascular failure gives rise to hæmorrhage. Where acute nephritis has left more or less œdema, there is, towards the end, a liability to hæmorrhages. There may be bleeding from the gums, bleeding from the nose, blood extravasations beneath the skin, blood in the urine. The patient may pass blood from the bowel, cough it up from the lungs, vomit it up from the stomach. Such hæmorrhage may be repeated until the patient dies. Here let me say that such hæmorrhages in the skin are not purpura, or, if we call them purpura, we must say, "purpura in connection with acute Bright's disease." Sometimes patients are taken into hospital for purpura, and then it is found that they are suffering from acute Bright's disease.

Now, here we have to ask, How comes it about that the blood and serous circulation thus fatally fail? To answer that question, we shall have to take a very wide survey. To lead up to the answer, let us first notice that, owing to the pathological view being confined to the œdema, most earnest efforts have been made to get rid of the dropsy, and so to restore the breathing, circulation, and nutrition, to healthy order again. For that end, hot-air baths, purging, and other measures, have, for many years, been resorted to. But it has been a frequent experience that, in extreme cases, these measures failed. In other cases the hot-air bath much diminished the œdema, and then convulsions supervened, and killed the patient. In other cases the purgatives failed at first to carry off the œdema, but after a time diarrhœa set in, and continued irresistibly, and then the dropsy entirely disappeared, but the patient died of exhaustion. We must not trust to purging for the cure of Bright's disease; under such treatment the patient gets weaker and weaker, and wastes, and the heart fails. I once knew a hospital sister who had symptoms of kidney disease for many years, but always refused to be treated by a doctor. "I've seen too much of it in the wards," she said, "Pulv. Jalapæ Co., every other morning, until the patient dies."

Experience has shown that such a limited view, considering the dyspnœa and other symptoms, including the inflammatory and uræmic, to be due to the œdema, is inadequate, and does not lead to successful treatment. It was right enough as far as it went, but was too limited, and it became necessary to go further and to look for the cause of the œdema. The œdema is demonstrably due to failure of the serous circulation, and for purposes of treatment we have to inquire how it is that the circulation thus fails. It is not merely due to the kidney being injured, for kidneys have been extremely decayed for months and years, and there has been little or no dropsy. In one post-mortem examination I found granular contracted kidneys, one weighing only three-quarters of an ounce, the other, two ounces and a half; this patient had been under observations off and on for ten years with symptoms of renal disease, and during the whole of that time there was no sign of dropsy. Such freedom from dropsy is a common experience in cases of granular contracted kidney, and where dropsy does arise in these extreme forms of kidney decay, it is either because acute nephritis has supervened, in which case we shall find, in greater or less degree, symptoms found with large white or mottled kidney, or, disease of heart has come on, and so the circulation has been weakened.

We have, then, to look beyond the mere kidney failure to ascertain why the attendant œdema is so prevalent in some cases and not in others. To learn the reason let us notice a guiding fact, that the dropsy is augmented with the increasing loss of red corpuscles, and that with the loss of these coloured respiratory organs there is increasing dyspnœa.

We are unable to confine ourselves to the view that this dyspnœa is merely due to the exclusion of air from the lungs consequent on the bronchial tubes becoming filled with fluid, for this is too limited. In some of these cases, where there has been the most extreme dyspnœa, so that the patient was gasping for breath, we have listened to the chest, and been astonished by hearing the breath-sounds distinctly. There were no moist or dry râles, no dulness of the percussion-note; we merely noticed that the breath-sounds were harsher and louder than normal, so that it was evident that the air passed abundantly into and out of the lungs. Again, in other cases there was much evidence of œdema of the lungs, and yet much less dyspnœa. In considering the shortness of breath, we must not, then, be guided only by the physical signs of œdema of the lung, for where there is undoubtedly much œdema, yet there may be adequate respiratory power in the blood to maintain the general circulation, because there is not extreme anæmia. Never let us overlook that it is not the "lung" we formally think of that breathes—not the pulmonary framework equally manifested by the corpse-lung—it is the blood circulating in the pulmonary plasma that breathes. What I wish you to understand is, that I have been led, by experiences of various kinds, to believe that it is the continuous transmission of energy from the red blood to the nervous and other tissues of the body that carries on inspiratory and expiratory proceedings, and that the bronchial tubes are thus kept free from fatal watery accumulations; but, as the blood pales with the onset of anæmia, the respiratory energy due to light is lessened and lessened in the serous fluids of the blood and tissues, until dropsy fatally accumulates.

We have contended that it is the air, moved by heat-energy, that lifts and carries on the water of the blood; that the rhythmical working of the heat circulates the watery vapour, and carries it out of the air-passages in expiration. But whilst there are many general experiences revealing that the breathing of human beings and of all other animals is dependent on heat-energy, we have now to consider that it is further dependent on light's rhythmical proceedings.

We cannot limit the consideration, and say it is merely the loss of heat-energy that makes the air and water of the serum incapable of travelling onwards, until the body becomes water-logged. Let us recall that removing the œdema by heat, where it was most extreme, has demonstrably, on the whole, been a failure, ending in death. We have been led to see that there is more than loss of heat-energy by noting that the œdema and anæmia—loss of colour in blood—are correlated. Nor can we be content by saying that the extreme loss of red corpuscles must be attended by lessened oxidation, and consequently the quickening energy be taken from the serous fluid until it stagnates. We cannot overlook that this is so; but we equally cannot overlook that the colour of the blood is lost.

We have therefore been brought to see that in the human circulation, as in the vegetable circulation, colour is significant of power—simply speaking, of immense power. We have to recall that colour is due to light, and that the blood owes its colour to light; and that the greatest activity of blood and the greatest activity of respiration, with its influence on the serous circulation and on general nutrition, prevail and are most manifested when light-energy is most powerful on the earth—in daytime and in spring. And it is beyond question that in anæmia there is failure of respiration, in consequence of the corpuscles of the blood being deprived of colour (light)-energy.

In plants it is manifest, and is admitted on all hands, that life and light work continuously together; both chemical activity and nutrition in plants are dependent on light. The vegetative functions in animal and in human structures are, we have been led to consider, equally dependent on light, which, in human nature, is Heaven's life-giving energy. Therefore, the serous circulation fails, and the nutrition of the tissues fails, and the blood and the vessels at last perish, when the body is deprived of that heavenly supporter.

The trees and plants spread their leaves to reach the light—the power of Heaven which raises them up; the animals rise with the morning, and feast and fatten in daylight; children grow in health and strength, and are most happy gambolling in the sunshine; old persons, looking for renewed health, rise early to enjoy the freshness and purity of the rising sun. In countless ages there has been testimony to the life-giving influence of the light. Regarding human energy in its most material and substantial manner, what is it but light-energy working in or through the blood, which is made up of the products of the world?

Further, if we trace the effects of injuries, severe shocks, such as fractured pelvis, burns, or other great irritations, which fearfully affect feeling (consciousness), until the blood circulation ceases, we observe that air is heard to be breathed freely into and out of the lungs. On examination of the lungs after death we have found that the failure of circulation was due, not to any obstacle in the air-passages, nor to consolidation, collapse, congestion, or œdema of the lungs. But such lungs are much drier and darker than normal. Here, also, we have been led to believe that this travelling of gases into and out of the blood is not enough to maintain the blood circulation, especially the venous circulation. We have to recognise that respiration is a fine rhythmical working, and that light is indispensable to its proper performance.

By such common experience we have been led to consider that the blood circulation is dependent on respiration, this on the working of light-energy, and whilst that continues there is life in us; that respiration and mind act in us as one, and that we feel and see by the light that is in us—we know by the light that is in us. Might we not therefore exclaim with the old manly voice of him who, loving the light and loving life, feeling the light to be his master, to be the energy and the mind that gave him life: "He set a tabernacle for the sun, which is as a bridegroom coming out of his chamber, and rejoiceth as a strong man to run a race."

It is this view, that the tendency to the onset of dangerous œdema increases with the increasing anæmia, which has led me often in the wards to warn you against taking too limited views about food in the treatment of these kidney cases. It has often been the practice to keep such patients on nothing but milk for weeks together, but I do not think that a useful line of treatment. By food we can renew and redden the blood, by food we can strengthen the breathing and circulation. With varied food, fresh from the earth, we can convey into the body the stored light-energy of the heavens.

Here let me add that, in conversation with different scientific observers, I have been brought to think that there is a developing conviction that light-energy, passing into various material media, may become converted into, and subsequently manifested as, electrical, heat, or other energy. Isaac Newton held that light was the source of all motor power, and those who now believe that all energy comes from the sun and other heavenly spheres must find it difficult to contradict that view. As medical men we must recognise that we cannot confine ourselves to chemical views, for chemists have to proceed to account for the affinities and combinations—nor are physical views sufficient for us, these must look for the source of energy, or they are barren. The biologist comes to inquire and see how things live, come and go, and enjoy existence. And however far-fetched some persons may consider this teaching that I have put before you, and although in the daily travail of our practices we may think it will not work, yet we have to take the world as it is, and I may sadly add, after many years of effort and labour, that I have found the making of homunculus and bottling him up to be a sorry business, and after all there was the devil to be paid. I speak thus to you who are about to undertake the cares and responsibilities of medical practice, because, as I remarked at the outset of these lectures, pathology has to teach the maxims of rational practice, so as to guide us to appreciate the relative workings of the various structures of the body.

We have now to go on and consider *chronic Bright's disease*, with *granular contracted kidney*. In approaching this subject I would ask you to let me stay awhile, and recall to our minds that our forefathers inculcated that we should always render honour where honour is due, and I can see that it is in the economy of Nature to do so, for if we do so we are helped in return and encouraged. Encouragement every person needs to gain success, and only those can guide who have been the road. Here I gladly acknowledge that Sir William Gull was, at Guy's Hospital, my clinical teacher: he first taught me how to examine organ by organ, until I could take a more comprehensive view of the whole, and in the course of years, when our minds were working together on the subject of "arterio-capillary fibrosis," with granular contracted kidney, Sir W. Gull often enabled me to gather much insight from his very great experience and manly thought and feeling.

Before going further it may as well for me to explain to you what we understand by a granular contracted kidney, this being a matter about which there is no dispute. Under conditions which we will presently discuss, fibroid thickening occurs in the connective tissue of the cortex and in the capsule of the kidney. This thickened tissue slowly undergoes contraction. Being irregularly distributed, as it contracts it draws in portions of the surface of the kidney here and there, leaving other portions projecting. These projections are the "granules," and such is a granular contracted kidney. If the fibroid change is very scattered, only a little here and there, and this little has contracted much, the surface is drawn in to form little pits here and there. If, on the other hand, there is a great accumulation of fibroid tissue, and its distribution is almost uniform, the drawing in has been so complete that the granulation of the surface has almost disappeared. Then the kidney has a white, exceedingly finely granular, bloodless appearance, and cuts tough ; the cortex is much diminished in size.

In our investigations Sir William and I were soon brought to recognise that the local condition—granular contracted kidney—must be regarded as an organic failure resulting from different causal conditions, and that the clinical course—the pathology—differed accordingly. For instance, in some cases the kidney disease had resulted from an attack of acute nephritis, and was mostly a local affection, the remainder of the body being comparatively healthy, and if no further nephritis ensued the patient continued about for years. Such a case was that of the man of whom I spoke before, who had granular kidney for ten years and no dropsy. He first came to see me at Victoria Park. He used to say, "I think my master will think I'm putting this on." There was albumen in his urine, but he did not look very ill. One of my colleagues once asked me if I did not think he was shamming. Eventually he died in the London Hospital of erysipelas of the head and face. One kidney weighed three quarters of an ounce, and the other two and a half ounces, but there was no hypertrophy of the heart and no vascular degeneration.

In other cases the clinical history was altogether dissimilar—there were symptoms of degenerative changes in the tissues of several other organs, some of these having evidently come on at about the same time as the renal changes, others having preceded or followed these. Moreover, the tissue degenerations in the kidneys and other organs had, on post-mortem examination, so much similarity that we felt that we were warranted in considering them as having a relation to one another. Further, in all these organic degenerations it was manifested by the aid of the microscope that the arterio-capillary system had undergone fibroid changes ; the adventitia and intima had become thickened by fibro-cellular formation, and the middle muscular coat was, in parts, much atrophied, whilst in other parts it seemed much thicker than normal. That thickened appearance Dr. George Johnson had previously discovered, and most serviceably called attention to; he considered it to be due to increased muscular tissue, indicative of hypertrophy of the muscular coat of the artery—bestowing on it an additional vigour. We, however, could not take such a determinate position; we could not decide to what extent the appearance was due to mere contraction, to what extent to growth of muscle, or to some adventitious morbid growth which had taken place amongst the muscle cells, or how much the appearances were due to swelling by œdema.

We experienced, as regards this muscular coat of the vessels, a similar difficulty to that met with by pathologists, as regards thickened, or so-called hypertrophied, hearts. To what extent is that seeming hypertrophy due to new muscle, to means for increase of contractile energy, or to what degree is it morbid growth, or merely the thicker appearance of muscle in contraction?

The vessels are channels in connective plasma, provided with specialised structures to promote the safe travel of the blood, and to co-ordinate the serous supply according to the wants of plasma and organic functions. It was manifest to us that as the adventitious coat had become thickened by morbid growth, the connective plasma extending therefrom had been consequently invaded, thickened, and converted into a fibroid material, which had subsequently contracted and destroyed the organic forms. By this change the convoluted tubules of the kidney, the liver-cells and tubules, the medullary sheaths and axis cylinders of the spinal cord, the heart's structures, those of the bladder, the uterus, the skin, the pia mater, and other organs, had been affected—some of these organs had suffered in one case, and some in another, and the organic changes had occurred in various degrees. We could not, therefore, but affirm that there was a wide-spread tissue degeneration in these cases of chronic Bright's disease with granular contracted kidney, having as its basis failure in the arterio-capillary system.

In the course of many years, examinations at the post-mortem table had shown us that the brain, lungs, liver, spleen, stomach, uterus, bladder, skin, &c., might thus be, one or many of them, much altered by fibroid thickening, and that with these changes the kidneys had in some cases undergone much granular contraction, but in other cases, whilst other organs had undergone the fibroid change, there was little fibroid contraction of the kidney, or seemingly, to naked-eye examination, none at all.

We therefore stated that this granular contraction of the kidney, this fibroid renal degeneration, was part of a general fibroid degeneration; that in the cases with these increasing degenerations we recognised clinically a cachexia, and that it, with the diseased forms, constituted a morbid state which we termed arterio-capillary fibrosis. We considered that this fibroid thickening in one or more of the organs imparted an augmenting hindrance, a stenosis to the healthy organic and tissue workings, until it obstructed the circulation to such a degree that it brought about a fatal ending. Moreover, we stated that our further clinical observation in such cases had increased our conviction that the kidney affection was not in all cases the primary disease, nor could the other organic failures be attributed mainly to the kidney disease.

In some of the cases the failure had begun notably in the brain, and such patients had suffered from increasing blindness and deafness, increasing giddiness, increasing sleeplessness. In others the failure was in the spinal cord, and the patients had cramps, weakness in the limbs, irritability of the bladder, and difficulty in getting the fæces away. In others it had begun more in the lungs, and had led to increasing shortness of breath, to emphysema and bronchitis, or to progressive wasting, taking more and more the form of phthisis. In others the failure had been at the outset connected more with the stomach, there had been early signs of increasing failure of digestion, and wasting of muscle. In others it seemed to have attacked the blood, causing fatal anæmia—this is one of the forms of fatal anæmia which comes on at about fifty years of age, goes on for about a year or two, and kills in the face of all treatment. We should not overlook that in many cases the fibroid degeneration early attacks the uterus, and puts on fibroid tumour form with its attendant risk of hæmorrhage. It may attack the testicle or the bladder, the latter leading to what the surgeon speaks of as atony of the bladder. Fibroid changes having taken place in the bladder, with or without much change in the kidney, but with the usual hypertrophy of the heart, lead to recurring bleeding from the bladder and to cystitis; the surgeon sounds and finds no stone, stricture, or tumour to account for the bladder symptoms—but there is a heaving impulse of the heart, with signs of dilatation of the left ventricle, without aortic valvular incompetency, but with thickened arteries.

In whatever organ this morbid change had occurred, we found the organ was liable, owing to the change, to take on acute inflammation, which might rapidly disable the patient, and there was also in some organs liability to hæmorrhages. We gathered that our efforts must be directed to warding off these acute changes, to warding off pneumonia, to warding off cerebral hæmorrhage, or hæmorrhage from diseased vessels in other parts—commonly epistaxis, but not hæmoptysis or hæmatemesis—or to warding off acute delirium, acute myelitis, and much more commonly nephritis, with its fatal tendencies, or to warding off gastritis, cystitis, orchitis and so on.

Here let us ask, What evidence we have that granular contraction of the kidney is due to heart disease. I asked myself the question many years ago, and I said to myself that if it were due to heart disease, it would be found after death from heart disease in a certain number of cases at all ages. On examining a sufficient number of cases it was found to occur at all ages, but the proportion of cases of heart disease in which granular contraction of the kidney was found increased the nearer we got to forty years of age. Now forty is the age at which, in the absence of heart disease, granular contraction of the kidney tends to come on. It appeared therefore that the venous congestion of the kidney tended to promote a premature fibroid change.

In leaving this subject, let me here repeat, to make the position clear, that the cases of granular contraction of the kidney may be divided into two groups. Firstly, there are cases associated with hypertrophy of the heart, diseased vessels, and more or less wide-spread changes in other organs. Secondly, granular contracted kidney occurs as a local disease, without any hypertrophy of the heart, and very little—perhaps no recognisable sign of—degeneration in other organs. The latter kinds of granular kidney would seem to be in many cases the product of acute nephritis, and that acute nephritis may be due to various causes—syphilis included. In passing, let me mention that I have seen extremely granular kidneys in a young man who was the subject of inherited syphilis. There were neither hypertrophy of the heart nor vascular degeneration.

It has become clear to us, from clinical, pathological, and anatomical experience, that granular contracted kidney must be regarded according to its correlatives. In medical practice we have to consider the source of the failure in health, and regard the case according as one or other organic disease is the main source of failure.

To conclude our study of Bright's disease we will consider *uræmia*, which is a frequent cause of death. Always associate uræmia with acute changes in the kidney. Do uræmic phenomena never occur in chronic cases without acute change? We cannot afford to be dogmatic about this, but we usually find that uræmia is associated with acute changes in the kidney. Uræmia is common in cases of chronic Bright's disease, but they are cases in which acute nephritis has supervened. This has a very important practical bearing, for we may be free from anxiety about uræmia in a case of chronic Bright's disease, as long as we ascertain from time to time that there is no acute nephritis, and when uræmia does come on we treat the patient for acute nephritis.

What do we mean by uræmia? We now mean merely certain clinical phenomena, but formerly the word had a different meaning. Literally, "uræmia" means "urea in the blood," and this name was given when it was believed that the clinical phenomena of uræmia were due to the blood being poisoned by urea. We now know from experimental evidence that there may be much urea in the blood without its giving rise to uræmic symptoms, and it is admitted by most pathologists that it is very doubtful if the symptoms of uræmia are due to the presence of urea in the blood. When it was seen that the presence of urea was not sufficient to account for the symptoms, it was suggested that they were due to earlier excretory products—leucin, tyrosin, xanthin, &c. It is almost impossible to conceive but that there must be much accumulation of such excretory products in the blood when the kidney is not performing its functions properly, but we cannot consider that such accumulation is the main cause of the symptoms of uræmia. I speak like this because we have had to practise according to these chemical views, and they have proved unserviceable.

What is the use of theory? We may call a theory true if it is of great service, and the test of a medical theory is that it guides to successful treatment. Looking back through many years we see that the chemical theory led to the practice of purging the patient severely for uræmia, and the result was for the most part failure. Before discussing another view as to the nature of uræmia, and the treatment based upon that view, it will be well to describe the clinical phenomena.

Commonly the disturbances take a cerebral or cerebro-spinal form. In many cases there is increasing failure in conscious manifestations, the patient taking less and less notice, becoming more and more drowsy, but not altogether losing consciousness. If we speak to him he opens his eyes, answers, and goes to sleep again immediately. This has been termed "the quiet stupor of uræmia." It is so quietly undemonstrative that it may easily be overlooked, but we must be sure to recognise it, as it commonly passed into fatal coma.

Motor disturbances are very common, and may take one of several forms. There may be restlessness, spasms, and twitchings, which it would be proper to call convulsive seizures without loss of consciousness. The motor failure may be manifested by movements which might be mistaken for chorea. I will tell you of such a case. I was called in consultation to see a young woman at Ilford. When married, a few weeks before, she was thought to be well. I saw her lying in bed with repeated twisting, jerky movements of the hands and arms, and her eyes were shut. Abruptly she opened them and said, "Hold your tongue, mother; I will tell the doctor about it." Next morning she was comatose, and died. It was found to be Bright's disease.

The twitchings may be followed by convulsions with loss of consciousness. The convulsion may affect one or both sides, but, very commonly, affecting both sides, is much more marked on one side than the other, and on this side paralysis is left after the fit, but passes off in a day or two, there being apparently no permanent brain lesion. This is known as epileptiform uræmia, and the attacks may be indistinguishable from epilepsy. It is very surprising to learn, in some of these cases, that the patient had been doing his work up to the time when he suddenly fell down in a fit. I was one day called to the Bank of England to see one of the cashiers in a fit. He was lying insensible and violently convulsed, and I was told that he had been doing his work as usual up to the time of seizure. He had Bright's disease.

There is an apoplectic form of uræmic attack, in which the patient falls down, becomes comatose, and dies in a few minutes, without convulsion. A woman went into a public-house close to the hospital, and asked for some beer. She was in in the act of raising it to her lips when she fell down insensible, and died before they could get her to the hospital. At the post-mortem examination I found granular contracted kidney, with recent acute nephritis—there was no cerebral hæmorrhage.

In another form of uræmia there is acute delirium, and the patient may be sent to a lunatic asylum. The delirium is very impressive. It may be simply a fussy, meaningless activity; the patient will get out of bed and fuss with the bed-clothes, and get in on the other side, and then repeat this. There may be monomania. A woman in the ward took up the idea that the medical officer was committing rape on her. Probably in this woman there was great sexual activity when well, and sexual feeling was aroused when a man came near her bed—these two things determining the form of her delirium. Uræmia may be manifested by symptoms of a typhoid kind—stupor, sordes about the lips, and extreme prostration. Uræmia takes the form in some cases of a most distressing wakefulness.

In other cases it may not take a cerebral form, but some other organ is attacked; the intestine for instance, and uræmia is manifested by severe diarrhœa; or it may be the stomach, leading to violent vomiting, which continues until the patient dies. I have known such a case mistaken for cholera, the collapse was so marked. The only other condition I need mention is the rapid loss of sight which sometimes occurs in uræmia.

Let me sum up these symptoms for you by saying that uræmia may be manifested by: 1. Vomiting or purging.

2. Quiet stupor. 3. Spasm and twitching—convulsion without loss of consciousness. 4. Muscular spasm stimulating chorea, 5. Epileptiform seizures. 6. Apoplectiform seizures. 7. Delirium or mania. 8. Sudden loss of sight. 9. Excessive wakefulness. 10. Typhoid symptoms.

In all forms of uræmia, except vomiting and purging, the tendency is to die by coma, but with the exception of the apoplectic form, all forms of uræmia are commonly recovered from, and are evidently in such cases due to temporary conditions. When I was a young man I used to give a very gloomy prognosis in uræmia, but now, unless there is coma, I always hold out hope.

What do we find on post-mortem examination after death from uræmia? During life we can often see by the ophthalmoscope that there is œdema of the retina, and after death we often find, as we might expect, œdema of the brain and spinal cord. The brain is wet and soft, but I have never seen actual breaking down. The spinal cord is also soft, and swelled with serous effusion. Both in the convolutions of the brain and in the spinal cord we occasionally see with the microscope minute collections of colloid, albuminoid matter and scattered leucocytes; it is also common to find exudations of red corpuscles. In the "Transactions of the Pathological Society" you will find some coloured drawings of sections of the spinal cord made from specimens shown to the Society by Sir William Gull and myself; in these you may see the accumulations of albuminous material around the vessels.

Besides the œdema there is another condition leading to failure of nervous function. The nerve centres are usually found very anæmic. Further, especially in association with granular contraction of the kidneys, we very commonly find chronic degenerative changes in the nerve-tissues themselves. Often there is fibroid thickening of the pia mater, the so-called milky pia mater; there may also be atrophy of the convolutions. With granular kidney and degeneration of the vessels there is a liability to cerebral hæmorrhage, and the symptoms of large hæmorrhagic effusions in the brain may so much resemble those of uræmia that the diagnosis is often difficult. On the other hand, where death has occurred from uræmia, we may find cysts, or other signs of the remains of hæmorrhage into the brain.

In the course of many years of experience I have been increasingly impressed that there are many local, cerebral and other, organic morbid conditions, making up the symptoms termed uræmia, of which the chemical view gave no adequate account; with large white kidneys there was general œdema and anæmia—anæmia and œdema of the cerebro-spinal centres, œdema of the lungs hindering breathing and circulation, leading to distension of the right side of the heart, and venous congestion of the nerve-centres—all these are potent conditions tending to arrest the cerebro-spinal functions.

It is well known that anæmia of the brain may give rise to convulsions similar to those of uræmia. "Uræmia" may be said to be due to failure of the circulation and respiration in the cerebro-spinal system, attended by œdema and other exudations swelling the structures—that there is also an excessive accumulation of excretory products we do not doubt.

In cases of granular contracted kidney there may be gross structural decay of the brain and spinal cord—and this may be of a kind which, in the absence of Bright's disease, is often attended by epileptiform or apoplectiform seizures, or by muscular-spasmodic movements without loss of consciousness, or the changes may be such as are attended by delirious outbreaks or dementia. We are led to think that such changes being present, varying in degree and in kind, the failure of kidney function further weakens the decaying structures until uræmia supervenes, especially when there is present at the same time much cardio-vascular and pulmonary disease; but we think that were the morbid changes in the brain and spinal cord present in more extreme degree, they would lead to the production of the symptoms, convulsive or other, independently of the kidney disease. Let me here remind you that in speaking of suppression of urine at the beginning of this lecture, I pointed out that this extreme failure of kidney function led to death without the onset of the symptoms of so-called uræmia. Consciousness was maintained almost to the last.

What I have told you will show you that uræmia is a much more complicated condition than books give any account of; and our treatment must consist in carefully feeling our way, making rest and warmth the great basis, and promoting the circulation and breathing. If the circulation will bear it we may purge, but we must be guided by the circulation. How far should we promote the action of the skin? Again, I say, be guided by the general circulation. In proportion as the circulation of the brain fails, the brain becomes much more sensitive and the mind more fearful. I have been led, by practice in the wards, to see that not a few patients with Bright's disease have been frightened to death with such a thing as a hot-air bath, for instance. I once thought that I was going to do a very clever thing. I had a patient who was uræmic and very anæmic, and I told the house-physician to get some oxygen made in the clinical laboratory, and to give it to him to inhale. The convulsions became more and more severe, and nearly killed him. Then I saw what a stupid thing I had been doing. What was the use of putting oxygen into the lungs when there were not corpuscles enough to carry it away? Whatever the explanation, the convulsions got worse.

My treatment of uræmia is now very simple: a warm bed, rest, free from all agitation, a little liquid food. I have been much encouraged to find that with such treatment the symptoms commonly disappear. The view that uræmia is due to circulatory failure is of more practical help than the view that it is due to poisoning. Do as little as possible, and do not frighten the patient to death.

LECTURE LII.

NERVOUS DISEASE.

Let us consider, first, what is the meaning of "nervous." We evidently mean a fine power that one thing has of corresponding with another. It is contended that all living things—plants and animals alike—are nervous. It has been a puzzling question how it is that animalcules, which we can watch moving about in a drop of water night and day, so rarely knock against one another. It is evident that they can correspond. How is it that these structureless bits of jelly, devoid of nerves, can correspond? We have come to see that every living piece of plasma is endowed with more or less nervous energy. The embryologists teach us that a nerve is merely an outgrowth of a piece of plasma which gets to the surface, and has the power of communicating with the great energies of the outer world, and in course of time, from such simple arrangements a nervous system is evolved.

Let us inquire how nervous energy is to be gained. At the outset let us notice that the great in-takers—the digestive and breathing organs, the carriers of the grosser energies of food, water, and air, which they transmit until they are united in the blood, by which these energies are to be distributed throughout the body—these in-taking organs are covered by the great sympathetic and vaso-motor nerves. It is evident that nervous power—the vis nervosa of which I have so often spoken—is placed in relation with these organs, to influence, and have at command when necessary, their activities. The brain, as is demonstrated by the process of its development, is a conglomeration, an aggregate arrangement of nervous tissues, surrounding an arrangement of vascular tissues, by means of which energies may be drawn from the blood. The nervous structures of the brain have been specialised in the course of development for specific aims and ends—optic, vocal, manual, and other. The structures of the brain and spinal cord are specially arranged as centralising communicators, and in each centre there are polarising cells, with extremely fine members extending in many ways. It is evident that these cells are means to receive, re-arrange, and re-transmit, in a more useful manner, nervous energy.

These ganglionic arrangements have two great methods of communication with the outer world. On the one hand, they receive sensorial impressions from the activities of the things of the outer world; and, on the other, they return—nervous energy passing into muscular working—constructive or destructive power into the things of the world again. There is thus, we are led to think, an interblending of the conscious energy with the energies of the world—these energies of Nature being the eternal operations of the One Great Mind of the One Great Worker and Maker of All. By this continued communication between the things of the outer world and the mind, the conscious activities within us, we are enabled to be really instructed and to be stored with nervous power.

Here let me say to you that there is much erroneous and inimical talking about the nervous. I can say that, for I have met with it, and have seen how others have suffered much from the misunderstanding. There is nervousness that distresses much, but it is owing to the fact that persons have been led to be too self-conscious. People will often say to you in practice, "Doctor, I've come to you because I'm so nervous," and you will have clearly to understand what they mean. They mean that they are unduly conscious of the correspondence with the outer world. They will tell you that they are afraid when people are standing behind them, that they are afraid when they are walking in the street for fear they should run against people. There is a necessity for the nervous correspondence, for it is by this that they are prevented from doing what they fear, but they are too conscious of the correspondence—they are bothered in the conscious. We must recognise the necessity for the nervous, but we must relieve the fear. This state occurs more or less in all illnesses, but especially in the beginnings of mental disease.

It really is a grand thing to be nervous. Often much unpleasantness and pain are mixed with it, but that is owing to abuse. The world's work is done by the nervous—we may see that the greatest workers are the most nervous, the most sensitive among men. Keener than the hawk amongst its kind, they vibrate from head to foot with nervous energies, and when seen airing themselves they help others immediately to take up a safe position for further progress—theirs is the "sovereignty of Nature."

Every healthy living thing is nervous, but must not be conscious overmuch, for this leads to intimidation. We are all of us subject to such intimidation. I remember after the epidemic cholera in 1866 I had very troublesome neuralgia in the shoulder. One man thought it was gout, another hinted it might be syphilis, another suggested bone-disease. I had myself seen precisely similar neuralgia in the shoulder in cases of aneurysm, and for months and months, while the pain lasted, I was intimidated by this fear of aneurysm. I am too old for that now: when I have pain I bear it as best I can, without thinking too much of what it may mean.

Impressions are received from the outer world by the scattered sense-organs of the skin, and in the special sense-organs there is provision made to enable us to gain further impresssions of the qualities of things—colour and form by the eye, workings of sound by the ear, sapient energies by the mouth, odoriferous energies by the nose. Thus feeling our way into Nature, into the Great Mind's operations, we become instructed as men. Opinions fade behind, like stepping-stones in journeying across the stream, and thus by the continued use of the senses come knowledge, joy, and power.

If any should tell us that it is the influence of sense that leads to sensuality, the answer is ready—What gift in the human body is not abused? We may reflect that it has been the wonder of ages that the universe goes delightfully onwards, leaving those that will to abuse and abuse, until they learn how to get sense and reason, knowledge enough—that is, power—enabling them to use in order to reach happiness and usefulness.

It has been demonstrated by experience that the impressions and energies received by the sense-organs of the skin are powerful aids in maintaining the breathing and promoting the general circulation—the nervous energies of the outer world transmitted through the sense-organs of the skin to the spinal cord, and thence reflected to the nervous plexuses governing the circulation and the breathing. In a similar manner this nervous power, derived from communication with the outer world, influences, both more directly through the sympathetic and other nerves, and less directly through the blood and serous circulation, the excretory function of kidneys, lungs, skin, and bowel, thereby co-ordinating and promoting the getting effete material out of the body. How indispensable to the proper maintenance of breathing and circulation is the integrity of the communication between the nervous system and the outer world through the medium of the sense-organs of the skin, may be learned experimentally by covering the skin of an animal with varnish. In such a case the circulation rapidly comes to a stop. In human beings we can see a striking demonstration of failure thus brought about when the skin has been extensively burned. The nervous communication with the outer world is cut off, the circulation fails, and collapse comes on. In such cases there are always the same morbid appearances; the left ventricle is firmly contracted and empty, and the lungs are lighter than normal, showing that the blood could not get through them,

On the other hand, whenever the breathing, the gaseous circulation, becomes difficult, the nervous working of the body becomes disturbed, and the mind is alarmed; there is uneasiness, more or less disease. We see this in all morbid conditions interfering with the proper interchange between the gaseous elements in the blood and the outer atmosphere. Where the nervous structures have undergone morbid changes, interfering with the co-ordinated nervous regulation of the breathing, the lungs become much congested, the air-cells filled with serum and corpuscles, and the pulmonary circulation is unable to continue. It is in this manner that death is brought about in cases of hæmorrhage into the brain, or other compression of the brain, and in cases of morbid growths in the cerebro-spinal system, with or without paralysis, and also in cases of acute ascending myelitis. Severe inflammation of the serous membranes leads to great irritation and disordered tension in the nervous working, and breathing is similarly affected. It is in this way that peritonitis and pleurisy usually end life. The finer operations of consciousness may become disordered, leading to restlessness, anxiety, and extreme worry in feeling, and this leads to much difficulty in the operations of respiration and breathing, until ultimately they may fatally fail. We see this in various epileptic and insane conditions.

There is a most impressive demonstration of failure of breathing in cases of shock from severe injury, to which I have several times before alluded. Immediately or very soon after the shock, the temperature of the body falls, the skin becomes darker as venous congestion comes on, the consciousness is clear, but increasingly feeble, the breathing becomes more and more labouring and struggling, cold sweats appear, then the mind no longer consciously manifests its presence, the pulse ceases, and breathing comes gradually to an end.

By such considerations we may in some degree trace how disease comes about. Respiration is everywhere operating in the body, and mind and respiration act as one; we can to a certain extent understand how it is that daily hindrances in the proper working of the nervous power, hindrances in the feeling, such as are popularly spoken of as worry, may interfere with respiration and breathing and general circulation in such a way, that, sooner or later, very deleterious effects are brought about in plasma, and especially in structures the nutrition of which has been previously weakened from inherited or acquired proceedings. Moreover, if the daily associations be such that nervous power is much weakened—made exceedingly unrestful by too much indoor proceeding in vitiated air—pulmonary function may become much enfeebled, and atrophy of the lung plasma and phthisical destruction may supervene. In other cases, with other associated conditions, the failure may occur in the kidney, the uterus, or other organs.

I repeat that we have to recognise, that if living creatures, and especially human beings, the most nervous of them all, are too much cut off from communication with the outer world, there is failure in nervous power, evidenced by weakness of circulation and shallowness of breathing. Herein lies the great risk of living alone—we often see in widows symptoms of such failure—feeble pulse, cold extremities, and shallow breathing. To many families the medical man is a great blessing in this, that he is a means to enable them to correspond with the outer world.

I think the best way of proceeding with the study of nervous disease will be, first, to describe hysteria, next, grosser changes in the spinal cord, and lastly, to consider brain disease.

Hysteria is a functional disease, distinguished by much disturbance in the cerebro-spinal system, taking the form of paræsthesia, hyperæsthesia, and in more advanced stages anæsthesia. With these disturbances in sensation there is more or less aberration of mental functions, notably of perception. As the spinal system fails greatly, motor disturbances supervene.

Hysteria is now admitted to be a very serious condition. It may cause more misery in a family than cancer, or other gross morbid change. It is a disease which frequently continues for years and years. The last time I was in the law courts about anything of this kind, was at Guildford, as witness in an action brought by a hysterical Jew against the London and South-Western Railway Company. The plaintiff's counsel asked the medical witnesses if hysteria were not a serious disease, and we had to admit that it was. It is, however, only through years and years of ignorance that medical men have been brought to see that hysteria is a serious disease.

I cannot speak of morbid changes in hysteria. When hysterical patients die, it is mostly from other diseases. The summer before last I was attending at the West-End a woman of my own age whom I had known for twenty years. For seven years she had been kept on her back by an hysterical affection of the spine. I was in consultation with two of the most distinguished physicians for mental disease in England. It was to me a most instructive fact that both of these men thought that she was more or less shamming, and that the cause of her disease was of her own making—"Drink." So to please them I told her to give it up, and she gave it up to please me. A few days after this she was driving in a carriage, became comatose, and died. She had carcinoma of the lung. The sputum and the physical signs had told me that long before, but she was thought to be shamming. You will, in practice, meet with many similar cases in which hysterical outbursts mask very serious disease.

Hysteria begins with increasing disorder of sensibility in the great sensitive nerves extending from the spinal cord to the skin—the sensibility becomes more and more morbid—there is weary aching, neuralgia, and the disturbance may culminate in very marked anæsthesia. The change may also occur in the nerves of special sense, leading to hysterical amaurosis, or hysterical deafness, or there may be paræsthesia of taste or smell. Partial anæsthesia of the larynx is not uncommon. Often there is anæsthesia of the lower part of the bowel, so that it will tolerate the accumulation of an enormous quantity of fæces; a similar condition may affect the bladder, but this is less common.

If our means of communication with the outer world are in a morbid condition, what can we do but feel morbidly? We do not feel the world as it is; we speak as we feel. Then what do we do? We lie. "They're such liars!" you will often hear said of hysterical patients, but the poor things only speak as they feel. Their minds are unhealthy; and it is difficult to know whether to call them insane or not, but delusions are very common, and even hallucinations.

When the sensibility of the body is healthy the breathing is healthy, but in hysteria the breathing is shallow and the lungs waste. The percussion-note is flat and expiration is prolonged, owing to loss of elasticity. "Is it phthisis?" you will be asked. Note that the change is equally marked in both lungs, and never let the patient or her friends think of phthisis until there are signs of consolidation. We have come to recognise that in madness all the organs of the body may be mad.

Healthy appetite and digestion, like healthy breathing, depend on healthiness of communication with the outer world. We are therefore not surprised to find that these hysterical patients commonly lose all desire for food and waste much. This condition has been called "apepsia hysterica." A marked difference between hysterical failure of digestion and that found with organic disease, is that solid food is usually digested better than liquid.

It is now recognised on all hands that movement is excited by feeling, and that feeling always precedes muscular movement. As the feeling of the hysterical patient becomes unhealthy, the movement also becomes curtailed and unhealthy. Then arise spasms and convulsions, and the so-called hysterical hemiplegia and paraplegia. This last is not paraplegia in the ordinarily accepted sense of the word; it has been well called "paraplegia from idea." They *think* they cannot walk. When that "I think" is moved they can walk; but so fixed is the idea in many cases that the doctor fails to overcome it. Hysterical paraplegia is distinguished from ordinary paraplegia by the paralysis never being complete. Further, we notice that there is greater difficulty in extension of the leg on the thigh than in flexion; whilst the reverse is the case in paraplegia due to organic lesion in the cord, in which voluntary control is not entirely lost. Hysterical patients can usually draw up the legs with considerable force, but are not able to extend them so well.

In hysteria, in association with the other disorders of sensibility, there is sometimes disturbance of the bladder; but as in other diseases of the spinal cord in which failure of function is not extreme, severe bladder disorder does not arise unless the disease is far advanced. There was an old physician who used to say, "An hysterical young woman will never wet the bed, or wet her clothes, until there is permanent organic disease of the cord."

Disturbances in phonation are very common. The so-called hysterical aphonia, indicating disturbance in the working of the pneumogastric, and other disturbances in the working of the same nerve, lead to hysterical vomiting. Both aphonia and vomiting are common in other diseases of the spinal cord and medulla.

Failure of general nutrition may be so great as to lead to extreme emaciation, and with the failure of nutrition the blood-vessels suffer and hæmorrhages are common. Hæmorrhage in the throat leads to the so-called hysterical hæmoptysis; small hæmorrhages occur, and there is coughing up of a little blood morning after morning, but the bleeding does not continue throughout the day. There may be hæmorrhages into the skin, and even sweating of blood, which used to be looked upon as miraculous. Instead of red corpuscles passing out of the vessels, there may be serous exudation leading to localised œdema, especially about the joints. In this point also hysteria resembles other diseases of the spinal cord.

During these varied disturbances it is usual to meet with storms on the mental side, and it often becomes difficult to decide whether we have to deal with hysteria or with mania. In such cases be slow to give an opinion.

Lastly, recognising these conditions to be the result of an early, possibly atomic, alteration, we are never warranted in pronouncing a hard-and-fast opinion as to curability. We must always be scientific enough to recognise that there is a real basis of tissue-disorder, and that the disease must therefore be treated really and sincerely. I have always made them feel that I was sincere.

Looking at the early conditions, we see that the cause of hysteria lies in not trusting the sensitive organs, skin and others, in their correspondence with the objective natural world. There is too much care, too much solicitude, and hysteria is commonly found in the mother's pet. She fails to see that, by her own excessive self-consciousness, she is rearing her child to be extremely self-conscious, and morbidly timid and apprehensive.

And the remedy—what is it? It sounds simple, but is really exceedingly difficult to practise. The remedy is—let them alone. I have been paid hundreds and hundreds of pounds, just for letting them alone. The patient's friends knew all about the disease—but I did not. I just let the patient rest, and guided her to be out of doors as much as possible.

The happiest thing to reach on the face of this earth is merely to exist—to live with the universe, and to feel that all is provided for—but we never reach that until we have got rid of many friends.

Diseases of Spinal Cord.—Let us now leave hysteria, and pass to consider grosser changes in the spinal cord. Morbid changes leading to symptoms of spinal cord disease may have originated either within the substance of the cord or extraneously. We will first briefly deal with some of the latter conditions.

Inflammation is very liable to extend to the membranes of the cord when there is severe inflammation in the vicinity. I have known suppurative spinal meningitis excited by inflammation in the pelvis, and travel right up to the brain. Any protracted inflammation of the bladder or uterus, even gonorrhœal inflammation, may extend along the nerves or the veins to the spinal cord. Bear in mind also that whenever there is severe inflammation within the abdomen or the chest, the spinal cord may be much affected; for instance, a man may have a severe pleurisy, and lose the use of his arm while it lasts.

One of the commonest conditions leading to inflammation of the cord by extension is caries of the vertebræ, the inflammation passing from the bone to the dura mater, and thence to the pia mater and the cord. In other cases there is caries of the ribs or of the clavicle, and inflammation passes along the intercostal nerves, and perhaps excites fatal suppurative meningitis. Inflammation excited by injury may extend to the spinal cord. Laceration of the cord is common in fracture of the spine, and it is important to remember that in the spinal cord, as in the brain, there may be much contusion of the nervous tissue, laceration of the membranes, and hæmorrhage, from a fall, a severe strain, or a blow, without any appreciable fracture of bone. This is common enough in railway accidents.

The membranes may be irritated or the cord compressed by some outgrowth within the spinal canal. Thus, an abscess, arising from carious bone, may compress the cord, so as to lead to extreme failure of function, without there being inflammatory change in the substance of the cord. However, some of the most gloomy cases of paraplegia have been from that cause.

Still these cases are amenable to treatment. Carcinoma, too, often compresses the spinal cord, or in other cases carcinomatous growth extends through the intervertebral substances, and involves the membranes of the cord. Hyperostosis may compress the cord: this is generally syphilitic in origin. Rheumatoid thickening may occur around the intervertebral substances, but this very rarely leads to compression of the cord. Enchondromatous, sarcomatous, or myxomatous growth may compress the cord.

You will be asked about *hæmorrhage* within the spinal canal. If there is blood between the dura mater and the bone, it is commonly due to fracture. In this region, however, there is a great system of veins, and if there has been extreme venous congestion we may find a little blood extravasated here and there. If blood is found between the dura mater and the pia mater, it has probably trickled down from the cranial cavity. If no such source of the bleeding could be discovered, we should suspect that there had been an aneurysmal condition of the small arteries supplying the cord, and that this had led to rupture, but this is a very rare condition. Hæmorrhage beneath the pia mater and in the substance of the cord is generally traumatic, but it may be due to some purpuric condition.

Syphilitic change, gummous growth, may affect the membranes of the cord. We find a yellow mass, surrounded by fibroid thickening, and the membranes are stuck together and to the surface of the cord. *Aneurysm* often erodes the vertebræ, but it is only in rare cases that it goes right through into the spinal canal. When this does occur there may be paraplegia from pressure, or the sac may burst into the spinal canal. *Pachymeningitis* is a slow extreme thickening of the dura mater, layer upon layer. It is supposed to be a chronic inflammation of the dura mater. It is especially liable to occur in the cervical region. It is usually accompanied by symptoms of dementia. If there have been no such symptoms, we suspect it to have resulted from injury. The course of the symptoms of pachymeningitis is very chronic. *Hydatid* may grow into the membranes, and lead to compression of the cord.

The advance of any of the above-mentioned growths may simply lead to pressure on the cord, and to signs of increasing weakness, but they more frequently act upon the nerves within the spinal canal. Pain is therefore a common symptom, and usually increasing pain. If the growth is localised the distribution of the pain is also localised, along one or two intercostal nerves, for instance. Signs of paraplegia generally indicate that the spinal cord is extremely compressed and softened. As the compression takes place there are disturbances in the reflexes, which are commonly at first exaggerated, and later lost.

We will next consider *spinal meningitis.* It may be due to an infectious disorder known as *epidemic cerebro-spinal meningitis.* Such meningitis is always purulent, and the disease ocurs in young subjects. On post-mortem examination we find no other morbid change but meningitis. There is no bone disease and no tubercle, but on cutting through the dura mater we see the pia mater covered with pus. The disease is endemic or epidemic in low damp regions, such as river beds, and we find it especially in association with great poverty. What is the actual suffering? The disease begins with shivering, vomiting, and pains which are not unlike those of rheumatism, with rise of temperature. The case is liable to be mistaken for rheumatic fever, but the shivering and vomiting should always make you suspect something more than rheumatism. In a day or two there is marked loss of power, and opisthotonos supervenes. Then we know there is spinal meningitis. Delirium comes on, there is marked paraplegia, and increasing loss of power over the bladder; the lungs become congested and the breathing labouring. Finally, death takes place from coma.

You may see somewhat similar symptoms in *meningitis due to caries*, but the pain is more localised round the body. It is well to recognise that such pain may precede any marked rise of temperature, and it should warn us immediately to put the parts at rest. After this localised pain, loss of power comes on. In some cases the pain becomes diffused, and we know that the meningitis has become general. In others, the pain remains very localised, but there is increasing paraplegia, and we suspect that the inflammation has gone through the substance of the cord and softened it. With the onset of myelitis there is, in addition to paraplegia, paralysis of the bladder and commonly also of the rectum. What other symptoms might we expect in meningitis due to caries? The disease is close to the pleura, and hence pleurisy is common. Inflammation may extend to the lung, and lead to suppuration, and a high temperature for weeks and months. In these cases of spinal caries our prognosis should never be extremely unfavourable unless the patient is actually dying. Recovery may occur after months and months of paraplegia.

A third form of *meningitis is due to tubercle*, and this is commonly associated with a similar condition of the membranes of the brain. There is acute cerebro-spinal tubercular meningitis. There is also a form of slow strumous meningitis—a chronic strumous deposit which may continue for many months, and the patient ultimately recover.

Acute myelitis is evidenced to the naked eye by softening of the cord. Under the microscope we see that the softening is due to a corpuscular and serous exudation into the connective tissue and nerve-tubes of the cord. We see groups of leucocytes here and there, and a quantity of hazy albuminous material which has swelled up the connective tissue. The medullary sheaths and the axis-cylinders are also swelled by the exudation. In some parts it is so abundant that it is difficult or impossible to detect the nerve-structures—they have either been destroyed, or completely buried up, in the exudation. We must not say that all softening of the cord is due to myelitis, as do some writers, for beyond question in some instances no inflammatory changes can be detected with the microscope—white softening appears to be a kind of necrosis, and corresponds to white softening of the brain. Acute myelitis affects all the columns of the cord, but especially the white matter, at any rate the change is more visible in the white matter. It may be very localised, or the inflammation may be widely extended throughout the cord, reaching even to the brain.

What are the causes of acute myelitis? (1) Injury, with or without fracture of the vertebræ. (2) Inflammation extending from without, especially in cases of caries of the vertebræ. Inflammation may also have extended along a nerve or a vein, or there may have been some other irritant, such as the pressure of carcinoma, aneurysm, hydatid, or other growth. (3) Strumous change, which may either be in the membranes or in the substance of the cord. (4) Venous congestion. If we examine the spinal cord after death from various causes, we are astonished to find how often there is exudation owing to venous congestion. Like the liver and the kidney, the spinal cord is very subject to venous congestion, and it is very apt to take on acute inflammation from this cause. (5) In some cases of acute myelitis or of acute softening of the cord, the symptoms come on in a very surprising manner. For instance, a young man may walk to his business, and suddenly in the course of the day find that he has lost the use of his legs. Cases of this class are probably connected with failing respiration and increasing fulness of the azygos and intercostal veins, leading to venous congestion of the cord, the cause of the initial failure being most probably extreme fatigue. It is notorious that extreme fatigue, especially if attended by mental shock, may give rise to acute myelitis. A ballet-dancer was on the stage of the Standard Theatre when there was an alarm of fire. She rushed off the stage to the top of the staircase, and then fell down, having lost the use of her legs, and was admitted into the hospital with acute softening of the cord. In other cases, however, neither antecedent fatigue nor mental shock have apparently been operative. I remember a patient in George Ward who had sat down to his Sunday dinner feeling quite as well as usual, but when he tried to get up, he found that his legs were paralysed. In these cases we are led to suppose that there must have been some insidious change in the substance of the cord, and then rapid exudation has occurred, swelling up the structures, and leading to immediate paralysis. (6) Acute myelitis may supervene upon sclerosis of the cord. In cases of sclerosis we should always be on the look-out for symptoms of acute myelitis, demanding that the patient should immediately be put at rest in bed. In all these fibroid changes in the spinal cord, as in fibroid change in the kidney, acute inflammation is liable to supervene from time to time, and is commonly recovered from. In other cases, as in pneumonia, or in acute nephritis, the antecedent fibroid change may be so extreme, and the vessels so much thickened, that the exudation may lead to complete destruction of the remaining functional portions of the tissue. (7) Toxic paraplegia, excited by alcohol, prussic acid, aconite, &c. How far in these cases the changes in the cord are secondary to venous congestion, is an open question. It is certain that all these poisons are such as produce venous congestion.

Whatever the cause, the actual suffering is much the same in all cases of acute myelitis. The brain is cut off from the lower part of the body, for the softened part can no longer conduct the nerve energies. Hence there is loss of sensation and of motor power, which soon becomes complete. As sensibility diminishes, the reflexes commonly become exaggerated. Softening of the cord involves the grey matter and the trophic centres of the cord, and hence sloughing is apt to come on. Wherever there is much pressure on the paralysed parts, sloughing soon comes on, and sores are formed over the sacrum, the heels, the toes, where pressed upon by the bed-clothes, &c., and the severity of myelitis may be estimated by the degree of sloughing. We should particularly notice that sloughing comes on early. There is paralysis also of the bladder, leading to retention, and in bad cases, suppurative cystitis soon arises. In relieving the retention, do not let us overlook that the urethra is anæsthetic, and that we may make false passages without the patient showing any sign of pain. For this reason we generally use soft rubber catheters, which do not permit of our using much force. The rectum is also paralysed.

The softening is usually in the dorsal or dorso-lumbar region of the cord, and the risk always is, if the patient does not die rapidly from sloughing or bladder disturbance, that the myelitis will extend upwards until the chest and the diaphragm are paralysed. In these cases the breathing gets slow and labouring; subsequently vomiting comes on. There may be a very irritating cough owing to failure in the larynx. Finally the heart becomes completely paralysed, and the action of the diaphragm ceases. These patients are singularly calm. They express no anxiety, they are not delirious, and it is only just at the last that they become comatose.

Like acute nephritis, acute myelitis is a recoverable condition. We are therefore guided in giving our prognosis, not so much by the degree of paralysis, as by the cause which has led to the myelitis. The prognosis will be most gloomy in cases of cancer, aneurysm, or hydatid. Where myelitis is due to bone-disease, struma, injury, fatigue, syphilis, or slow changes in the cord, the prognosis should be favourable.

Syphilis may excite myelitis, either by a gummous growth in the membranes, or by growth within the cord.

As regards embolism of the cord, I have never seen it, and it is admitted on all hands to be so exceedingly rare that for practical purposes we know nothing about it. Thrombosis also is very rare in the vessels of the spinal cord. There has of late been a disposition to account for white softening of the cord by assuming it to be a sequel of thrombosis. This is only an hypothesis. If asked about white softening, think mostly of acute softening without acute myelitis. What has been called "chronic softening," is usually sclerosis, with supervention of acute myelitis. Hæmorrhage within the spinal canal, not traumatic, and not due to cancer or aneurysm, is exceedingly rare. Growths may occur within the substance of the cord similar to those affecting the membranes, but they are rare. Syphilis and struma are the most common, but we may find myxoma, or sarcoma.

What do we mean by *sclerosis of the cord?* If I had not seen the appearances, I should be much confused by what the books say on this subject. To dismiss terms, I may tell you that the condition has also been called "overgrowth of the connective tissue in the cord," "fibroid substitution of the cord," "non-inflammatory hyperplasia of the connective tissue of the cord," and "chronic myelitis." On the Continent there is a disposition to regard these fibroid changes in the spinal cord, the liver, the kidney, and other organs, as chronic inflammation, and that is why this has been called chronic myelitis.

The fibroid change is always most abundant where connective tissue is normally most abundant. Hence it starts on the pia mater, and extends along the connective-tissue septa of the cord in towards the grey matter. The strands of connective tissue become much thickened. We may find a spindle-celled growth; but usually the change is more slow, and we find felty fibroid material. Commonly also the change extends from the grey matter, especially in the lateral columns. It is very marked about the arterioles, and very marked too about the nerve-roots, just where they arise. This is strikingly the case in *ataxic locomotrice.* The change may also extend along the anterior roots and into the anterior columns. If there has been wasting of the ganglion cells of the anterior horns of grey matter, such as occurs in progressive vascular atrophy, we may expect to find associated sclerosis in the anterior white columns of the cord. As the connective-tissue overgrowth increases, it spreads into the medullary sheaths. It destroys the white substance of Schwann, and, contracting, destroys the axis-cylinders. The process is very similar to that of fibroid change in the kidney. Owing to the destruction of the axis-cylinders, the lower extremities become weaker and weaker.

In speaking of granular kidney, I said how surprising it was that the patient might keep about for years, while the urine showed very little evidence of the change, so long as there was no acute nephritis. We see the same thing with sclerosis of the spinal cord. I remember a man who was taken into George Ward—I forget what for—and no one suspected that he had anything wrong with his spinal cord, until the sister of the ward complained to the resident medical officer that the man was so dirty in his habits, passing urine and fæces in bed. There was extreme sclerosis of the cord, with acute myelitis intervening.

Our prognosis when such acute inflammation supervenes in cases of sclerosis must be guarded, seeing that if the exudation passes away, the patient may be able to walk about again. It is not uncommon in slow failure of the spinal cord for rapid paraplegia to come on, due to a temporary weakness in the cord, and for the paraplegia to disappear as the circulation gets stronger. I remember Dr. Mercier once took me to see a middle-aged woman who had rapidly become paraplegic. She had been previously weakened by various conditions, so I gave a favourable prognosis. In a few weeks she was about again. As the blood-tension in her spinal cord increased, its nutrition improved.

Sclerosis may be general and wide-spread throughout the cord—a condition which may be compared to cirrhosis of the liver. It may be distributed in small islands, a condition known as disseminated sclerosis. It may affect certain well-defined tracts in the cord. There may be descending or ascending sclerosis, a secondary fibroid change, due to paralysis, on the one hand in the brain, on the other at the periphery.

Lateral sclerosis is a continuous widely spread fibroid thickening in the lateral columns of the cord. When paralysis is coming on we first notice trembling, then cramps, and later, rigidity, leading to the so-called spastic gait. There may be more complete loss of power, culminating in absolute immobility, but this is generally in cases in which acute myelitis has supervened.

In *disseminated sclerosis* there is weakness of the legs and tremor. The grey matter is little involved, and trophic actions are not much interfered with. There is no paralysis of the bladder and rectum, unless acute myelitis supervenes. The posterior columns being little affected, there is not much disturbance in sensation—we merely see exaggerated knee-jerk and ankle-clonus.

Ataxic locomotriee is another form of sclerosis, affecting chiefly the posterior columns, round the nerve-roots. Some describe the change as chiefly limited to the nerve-roots, and further, in some cases there has been evidence of neuritis at some distance from the cord. Hence the question has arisen, whether the disease may not be primarily due to a neuritis which extends to the posterior roots, and subsequently to the posterior columns. What is the suffering? At first there is disturbance in sensibility. The patients feel as if they were "walking on wool;" they suffer from "lightning pains." These, and other disturbances in sensibility, may precede the onset of the ataxic gait by some years. Loss of power of co-ordination is always more marked when the eyes are shut. The bladder suffers little, only towards the end; and there is very rarely paralysis of the rectum, but there may be some degree of constipation. The failure of sensibility leads to failure of muscular movement. It is in this way, not by direct interference with voluntary impulses, that the ataxia arises, and since the failure of sensibility is always more marked in the conditions of fatigue, rest should be a great factor in the treatment. It is simply astounding, sometimes, to see the influence of keeping an ataxic patient in bed for a few weeks. When the patients come into hospital they stagger, and perhaps fall, after a few steps; but with a few weeks' rest the power of walking much returns. It is wonderful what they can do if the consciousness of muscular action returns.

Descending and ascending sclerosis — how do these arise? Whenever the functional activity of nerve-tissue is much impaired or destroyed, the neuroglia becomes slowly thickened —this change may be seen in nerve-tissue throughout the body. The most common destructive lesions in the brain are in the Rolandic region, and when this region is grossly affected, the function of the fibres of the pyramidal tracts is in abeyance. A fibroid overgrowth takes place in the connective tissue extending along the nerve-fibrils which descend from the damaged area down the pyramidal tracts. Descending sclerosis is not visible to the naked eye unless it is very extreme. When we do see it, the affected regions have a bluish-grey appearance. In ascending sclerosis the lesion is at the periphery, and sclerosis extends upwards along the root-zones of the posterior columns, and along the columns of Goll and the direct cerebellar tracts.

In recent years we have connected *progressive muscular atrophy* with wasting of the ganglionic cells in the anterior columns of the cord. It is called also polio-myelitis. But we must remember that in progressive muscular atrophy in adults there is sclerosis as well as wasting, and it is an interesting fact that in descending sclerosis, producing perhaps symptoms like those of lateral sclerosis, symptoms of progressive muscular atrophy may supervene, because the anterior grey horns have become invaded by the fibroid change.

In these various affections of the spinal cord, and especially in *ataxic locomotrice*, there is a liability to the onset of joint affections—changes resembling chronic rheumatoid arthritis. There is a liability also to trophic changes in the skin, herpes and other inflammations, and even sloughing. There is a liability to trophic changes in the bladder. There may be outbursts of high temperature. There may be catarrhal conditions of mucous membranes, especially of the stomach and bowel, leading to diarrhœa and vomiting. Flatulence and gastralgia are very common. There is a liability to harassing windpipe cough, and also to pneumonia, which is a common cause of death in spinal cord disease. Such changes are especially common where sensibility is much affected, and we should therefore be on the watch for them in *ataxic locomotrice.* Lastly, let me tell you that the fibroid changes may extend up to the brain, so that symptoms of brain trouble supervene—giddiness, delusions, mental aberration, &c.

Diseases of the Brain.—In studying brain disease we have to bear in mind that there are two sides to the brain—structure and circulation. The one is formed, more or less fixed, and dead; the other is formless, undefined and living. In the struggle for life, we have to do our utmost to prevent these from being divided, for in the tendency to separate the circulating side from the fixed side lies disease. We must learn not to regard nervous disease too much from the formed side, but to feel the pulse and estimate the breathing and to give encouragement accordingly. In the treatment of brain disease what we have to keep before our minds is how to promote the easy working of the brain.

We often meet with demonstration of the fact that if the sense organs are interfered with too much, and the circulation in them interrupted, death results. Death may occur from pain, and on post-mortem examination we find no gross changes, and even microscopic appearances are doubtful. I once saw a man who had been caught up by machinery and whirled round and round. He was brought to the hospital, rapidly became comatose, and died. In fainting we have an example of sudden hindrance to brain function. Some people faint at the sight of blood—it is due to surprise, to the sight of that to which they are unaccustomed. With others, fainting is due to a peculiar smell; and others always faint when they take certain things into their stomachs.

When the blood circulation in the brain is failing, such severe symptoms of brain disease may arise that it is often a question whether there is a tumour in the brain. When the heart is failing it is common to get severe pain in the head, giddiness, mental distraction, and epileptiform convulsions, ending in coma. In other cases the failure of circulation in the brain is due, not to heart failure, but to deficiency of red corpuscles—there may have been a great loss from hæmorrhage, or the red corpuscles may be failing and dying. In such cases of anæmia we often have to ask ourselves, when severe symptoms of brain disease arise, whether there is anæmia because there is a tumour in the brain, or whether the brain symptoms are due to the anæmia.

In whatever way anæmia of the brain comes on, it is characterised by uneasiness and increasing distress in the head; there may be much pain—neuralgia, so-called; mental power is diminished and disordered; there is restlessness, which may pass into spasms, muscular twitchings and convulsions. Anæmia of the brain is attended by so much loss of healthy feeling that we are never surprised to find the mind become more or less "insane." There is increasing difficulty in thinking, and the muscular activities become very irritable, and, maybe, so uncontrollable that many such patients have to be sent to an asylum. In its extremest form it ends in coma. Dr. Savage has often impressed upon me the importance of recognising the relation between anæmia and insanity.

There may be a local failure of the circulation, a large effusion of blood pressing on the brain, causing loss of consciousness, which returns when the pressure has been relieved by trephining. In other cases there is no large mass, but some accumulated material on the surface renders the sense communications impossible. It may be lymph, blood, pus, or tubercle. In such cases the brain disturbance may also be manifested by convulsions. It will here be well, before going on to the study of the dead side of the brain—the morbid anatomy—to consider somewhat fully the nature and origin of convulsive seizures—of epilepsy.

Epilepsy and Epileptiform Seizures.—A man may die of nervous disease, with many marked symptoms, showing that the cerebro-spinal functions are much interfered with, until they fatally fail; we are bound to acknowledge that the cerebro-spinal organs were not working easy, were in fact extremely diseased, and yet after death we are unable to find any morbid appearances. That is why I take up the position that disease means loss of ease, because I have been led to see that a person may be killed by loss of ease, and we shall find no morbid appearances after death. Now that is the case in epilepsy.

When I want to test whether a student has a fundamentally clear view of pathology, I ask him this question, "What do you find after death by epilepsy?" In asking that question I want to see if he is a mere formalist who has got certain fixed ideas in his mind, or if he is a man of business. I want him to answer, "Nothing."

For many years post-mortem examinations have been made in cases of epilepsy to find out the cause of the disease, and all observers have been impressed by the absence of anything that could give a clear view as to the adequate cause. In some cases in which there had been fits there was thickening of the inner table of the skull, or an outgrowth of bone, in others some tumour had formed on the pia mater—a fibroma, perhaps; in others there was thickening of the membranes, but in other cases the membranes were not thickened, and in these also there were epileptic fits. They searched the brain and they found some slight changes here and there. At one time it was thought the mischief was in the medulla, at another that it was high up in the hemisphere. In other cases there was gross cerebral disease, a blood-clot in the hemisphere, but it was difficult to connect the clot with the epilepsy. There was this impressive fact, that in some cases in which there was the most extensive blood-clot there had been no convulsions. In one case a clot had ploughed up the hemisphere, and the blood had wandered down as far as the pons, and there had been no sign of epilepsy, whereas in another case in which there was a small clot situated near the pia mater, the disease had been ushered in with convulsive seizures like epilepsy. The same was seen in connection with abscess of the brain; it has been astonishing to find that an abscess may lie comparatively latent in the brain, and the man may go about in the open street, able to take care of himself, whereas, perhaps, in other cases one or more small abscesses have led to repeated convulsions.

I will give you another typical case. We cut across the brain horizontally, vertically, obliquely, in many ways, and by far the most of the brain was free from morbid appearances, and yet the patient had had repeated epileptiform seizures. We found, however, some syphilitic thickening of the membranes. We have to ask how it came about that with this comparatively small area of disease, while the great bulk of the cerebro-spinal system was apparently free from disease, the patient was killed?

A patient was brought into the receiving-room, and there was a history that he had fallen down in a fit. Perhaps it was epilepsy, but it was hinted that he was drunk, for he was seen to reel, and he used rather abusive language, and then became insensible. In the receiving-room, in the presence of learned opinion, it still appeared that he might be drunk, stupidly drunk, and so electricity was applied. Some rather sharp shocks were given, and they certainly shook him, for he turned round and swore lustily at those in the room. But he rapidly became insensible and died, and at the post-mortem examination I found meningeal hæmorrhage. With the exception of the blood on the surface of the brain the parts seemed to be healthy.

I will give you other cases. A patient in the hospital with heart disease gets weaker and weaker until he dies, but just before he breathes his last his body becomes violently convulsed; he has an epileptiform seizure. Or it may be that in the spasm of whooping-cough, as inspiration is interrupted, the blood is cut off from the brain, and there are repeated epileptiform seizures. We may see the same phenomena in laryngismus stridulus, or in a case in which a foreign body is impacted in the windpipe. As the circulation of the brain fails to an extreme degree epileptiform seizures are common. When the carotid arteries in a dog were tied the same result followed; the animal fell down in a fit. It makes no difference in which way the failure of circulation in the brain is brought about; it may be that the blood is cut off from the arteries when the heart is failing, or it may be dammed up in the veins because it cannot get through the lungs; in either case repeated epileptiform seizures may take place.

In another case the patient becomes paler and paler, often complains of pressure in the head, and of aching pain, and of a muddled, confused feeling; he cannot think, and becomes utterly indifferent to what takes place, and one day falls down in a fit and dies, and we find only anæmia of the brain. It is not uncommon for extreme anæmia to kill by epileptiform seizure. I have seen the same thing in Bright's disease; the patient gets paler and paler, and yet keeps about, and then suddenly falls down in a fit, dies, and all we can discover is that the brain is extremely pale. We say it was "uræmia," but that means there was poison in the blood. If there was poisoned blood running continually through the brain, how was the patient able to go on walking about, and why did he suddenly fall down in a fit? The blood was doubtless more or less poisoned, but the convulsion and death were due to failure of the circulation through the brain.

Anæmia of the brain means that the oxidation of the brain is failing—failing breathing power of the brain—and when this has become exceedingly feeble, if something comes to interrupt the continuous oxidation, if it is only for a moment, there may be a sudden loss of consciousness leading to death. While we recognise that the red corpuscles are the most important constituent of the blood, we must remember that if any of the other constituents of the blood are extremely diminished, cerebral function is much interfered with.

I remember one day one of the surgeons of St. Thomas's Hospital was talking to me about an attack of cholera he had. "I well remember," he said, "all of a sudden having a large evacuation, and I never felt so cleared out in my head in my life." The rapid withdrawal of water from his blood nearly made him lose consciousness. In what we call cholera sicca there is a rapid pouring out of water from the blood into the intestine, and the patient falls down unconscious, and dies before the bowel can empty itself externally.

Cerebral function is enormously dependent on sufficient food, and when I have to restore healthy function to the brain, I think chiefly of how it is to be fed. In the treatment of lunacy we have become very much alive to that fact.

When you are in practice you will be much impressed by noticing how much some epileptics are influenced by sound. I know of nothing that casts a more mysterious awe on my mind than this, when I listen to such patients, and learn from them that when they hear certain sounds they become livid, and may immediately pass into a fit. Sometimes while a patient has been talking of this to me the very thought has brought back the fear, and his lips have become livid.

It is a most remarkable fact in the history of the world that some of the most powerfully minded men have been subject to epileptic fits.

I used to go groping about in the bodies of epileptics, and trying to find out in that way the cause of epilepsy, but in that way I got more and more puzzled, until at last I thought I would give that up, and go and stand by the side of the living patient and listen to him. I thought "I will sit by his side as quietly as I can, and let his feeling teach me." The more I did that, the more interested I became in their stories. With them there is a wonderful struggle between darkness and light. When I see their lividity, the blood getting darker and darker, with increasing blueness, I am reminded of what Ruskin teaches about the belief of the Greeks that blue was no colour. I have stood by an artist, and heard him say, "The difficulty in dealing with blue is to prevent it getting too cold—blue is such a cold colour."

I stood by the side of an epileptic patient the other day, and she said, "I was so terribly bad yesterday; I had got the blues." I thought, "This woman is no fool; she means, 'What I want is red.' She feels blue and cold—cold right through her frame."

We seem to begin to see what blueness really means—heat-energy failing, light-energy failing. In my last lecture I spoke of the way in which light, consciousness, respiration, and breathing are continually acting as one. I am more and more brought to see that what we recognise as lividity, as blueness, is no colour, but a shade stealing right through the body, heralding the darkness of death. In epileptics lividity is a sign of all others we have to take notice of.

Now let us take another view of epilepsy. You will be asked, "What is the distinction between an epileptiform seizure and an epileptic fit?" The answer should be that a man hitherto healthy may become subject to fits, in form like epilepsy. It is often impossible to distinguish the outward form of the fit from epilepsy, but there is this great difference, that epileptiform seizures are due to some recently developed gross disease of the brain; it may be syphilitic or other growth, or it may be some change following a blow on the head. You may further be asked, "Do you mean to imply that a man of large experience, and one who has specially studied the subject of epilepsy, would not always in a given case be able to say whether the convulsions should be regarded as being epileptiform, or due to true epilepsy?" Answer, "It may be impossible to give a confident opinion—a man may fall down from cerebral hæmorrhage, from tumour formation in the brain, from Bright's disease, from obstruction in the windpipe, from aneurysmal or other intracranial pressure, and it may be impossible to say definitely that the case is not one of epilepsy." I wish you to be precise and dogmatic upon this point, for such has been the lesson of experience.

What are we to recognise as epilepsy? By this term, strictly speaking, we mean that there is a morbid condition disposing to fits from childhood upwards. The fits may be absent for weeks, months, or years, but in the intervals there are symptoms which we recognise as connected with the epilepsy—or, perhaps, it would be better to say, with the epileptic state. We can speak of something that is standing firmly and stationary, the epileptic state—we believe that there is some permanently stationary source of weakness in the tissues of the body; we have been brought more and more to see that the affection is not one of the brain alone, but also of the tissues of other parts of the body. Storms may take place, not in the brain, consciousness remaining unaffected, but in the heart. There are recurring violent attacks of pain about the region of the heart, spreading right across the chest; the pain is so violent that the patient has great difficulty in drawing breath, he becomes covered with a cold, clammy perspiration, his pulse becomes very small, and he is threatened with rapid death by collapse. "But is not that angina pectoris?" you will ask. Yes; but it is angina pectoris occurring in a person who has had epileptic seizures, and running a course like that of epilepsy, the fits recurring again and again for years. You will find instances like that recorded by Trousseau—one in which the angina appeared to be associated with *petit mal*, but later there were well-marked epileptic fits.

In another case it is not the heart which is affected, but the stomach. There are attacks of pain characterised by suddenness and severity, and accompanied by blueness and coldness, the patient saying, "I feel that if this goes any further I shall have a fit," and a fit may supervene. In other cases there is simply recurring gastralgia, with momentary giddiness. The pain may not be in the stomach, but pervading the abdomen, accompanied by diarrhœa, or by most troublesome constipation. In other cases there is pain in the face, violent attacks of neuralgia in the fifth nerve, the patient becoming blue and cold, and saying, "The pain is so severe that I feel I shall lose my senses."

I do not know of any part of the body that may not be affected by such attacks of pain in those subject to epilepsy. Pain in the eyeball may be very distressing, but those I have previously mentioned are the parts most commonly affected. I have been impressed in these cases to hear of pain, now in one part of the body, now in another. So often have I met with it, that now, when talking to an epileptic patient, I usually expect to hear an account of such attacks. One woman I remember complained of attacks of pain in the back, saying, "Do you think it is my womb?"

In other cases it is not pain which reveals the immense disturbances that have taken place in the conscious workings. Pain, let me say in passing, as a rule means fixity. When you think of pain think of fixity. It may be a thorn sticking into the hand, it may be a burn affecting the skin, it may be that a word has pinned a person down—in some way a morbid fixity has come about. The frightful disturbance in conscious working, instead of taking the form of pain, may take the form of a man rushing to find pleasure. Some years ago I took up a well-known translation of Dante, and read part of the preface. I read with great interest, for Dante's mind was one endeavouring to see into the struggles of poor self. It was pointed out, and I was much impressed by it, that there had been much experience in the history of mankind of endeavour to work the world entirely without pain; and on the other hand, there had also been much experience of endeavour to work it entirely in pain, pleasure being utterly denied. And both ways alike, being one-sided, resulted in frightful abuse. Some considered that the world was a very bad world, and that human nature was the worst thing in it, and that therefore it must be tortured and pained and denied and starved. But the application of these principles led to the outbreak of the most foul sensual abuses. Others said, " Why in this world should we be miserable? Let us eat and drink and dissipate, and let us get saturated with joy!" We know what happened then. I remember a woman (it is with no sense of finding fault that I speak grimly of these things), but this woman had shirked and shirked, and had not gone through the certain amount of struggle we must all go through if we would be strong and fair. She said to me, "I was in a railway collision, and they took me into the Leeds Infirmary, and they found I had got fracture of the collar-bone. And I cannot tell you what a relief it was to have real pain!"

We must not try to work with only one side of human nature. There are two hemispheres in the cerebrum, two sides to the pons and medulla, and for health, both sides must work together evenly. If one side is worked excessively, it may become enfeebled, and a terrific outburst of one kind or another occurs on the other. Well, that is very much how epilepsy comes about, as we trace it from childhood up.

In epilepsy we have to do our utmost to keep the circulation of the brain working evenly. Day by day, as we watch epileptics, we are impressed how peculiarly set they are, in their ways of looking at things, and of doing things. We see that this setness of their thoughts and actions hinders breathing and circulation more and more, until at last the patient falls down in a fit.

If you are ever asked about epilepsy by an examiner, think to yourself, "Now, does he want to know about the form of epilepsy? If so, I will talk to him about the morbid appearances found after death. I shall really be talking about nothing, but still I must say something, so I will say what has been said before. But if he wants to know about the treatment of epilepsy—how the patient is to be relieved, fits prevented, and a cure brought about if possible—then I will not speak of morbid appearances, but of suffering, and of how the patient is to be eased onwards, and to be helped through these 'sets.'"

I have seen the utter futility of much of this trying to cure epilepsy by keeping my mind dwelling too much on the anatomical side, and I try now to bring about a mutual thorough confidence between myself and my patient. When I have put aside morbid appearances, and reached that, I have been much more encouraged, and have met with much more success ; but this takes time to accomplish.

Affections of the Surface of the Brain.—It will help us in our study of the diseases of the brain, if we bear in mind that the circulation on which the function of the brain depends proceeds from the surface inwards; this being so, the greatest sensibility is at the surface, because there are the greatest activities. As the surface circulation is interfered with, the cerebral function must necessarily be lessened, until it ceases. Further, bear in mind that in the circulating blood are many powers—water, solids dissolved in the water, gases, corpuscles in suspension, albuminoids, heat, light, electricity, finer and finer operations. These combinations must not be overlooked, for we are now going to study disturbances in consciousness and other symptoms of brain disease. If any one of these energies is deficient or disordered in the blood, there are symptoms indicating that cerebral function is affected—if the water be lessened, as in severe diarrhœa or in cholera; if the gaseous elements be abnormal, as in very hot, ill-ventilated rooms; if the albuminoids are diminished through want of food ; if the heat energy is diminished, as in very cold weather, or the light, as in the dark days of winter; or if there be electrical disturbances—in one and all of these conditions, cerebral symptoms may be observed.

Except at the periphery, the tissue of the brain is not very sensitive; that is witnessed in cases of hernia cerebri, where the surgeon may cut away the protruding piece of brain with little or no feeling to the patient. It is notorious that a large hæmorrhage into the brain-substance may occur without causing pain; whilst hæmorrhage is occurring, the patient may be so little disturbed, that his night's rest is unbroken. Our experience is similar, as regards pain, in cases of abscess of the brain.

The surface of the brain is sensitive, and when there is pain associated with morbid accumulations in the interior of the brain, it is usually due to stretching of the pia mater. The body generally; and its organs, are sensitive at their surfaces. It will be useful to consider the diseases of the brain in two classes—those in which the surface is chiefly involved, and in which, therefore, pain is usually a marked symptom, and secondly, those chiefly affecting the interior of the brain, in which pain is absent, or less marked.

All diseases of the surface of the brain are characterised by pain, restlessness, and often other disturbances of feeling, such as vertigo, and commonly by vomiting, delirium, and spasmodic movements in greater or less degree; these may take the form of trembling and shivering, maybe like to ague, or convulsions. As I said earlier in the lecture, all forms of morbid irritation of the surface of the brain may produce an epileptiform convulsion, which is indistinguishable from an epileptic fit. Paralysis is not a symptom until the substance of the brain becomes involved, either by compression or by extension of inflammation, but in a longer or shorter time the symptoms of motor irritation and sensory disturbance commonly pass into paralysis and coma.

One of the commonest causes of surface affection of the brain is *injury*. Never let us overlook that a blow on the head, with or without fracture of the skull, may excite inflammation of the surface of the brain, and for a while it may be exceedingly difficult to make a definite diagnosis. Numbers of times it has happened that persons thus injured have been sent away from the hospital after the scalp wound has been examined and dressed, and have returned later with symptoms of inflammation of the membranes of the brain. I may remind you that a blow on the head may crack the skull without any appreciable displacement of the bone, and that the crack may be overlooked even on post-mortem examination, unless the bone be very carefully examined. A knock on the head may cause laceration of the membranes, and contusion of the surface of the brain, with more or less effusion of blood, leading to much disturbance, but the patient may ultimately recover. Injury may lead to permanent thickening of the membranes. It may, as said, excite suppurative meningitis and kill the patient. It may excite slow suppurative changes in the brain substance, and, months after the injury, an abscess is recognised. Any of these conditions may arise with or without fracture of the skull; more commonly there is fracture, which may be depressed, and therefore obvious, but it is very commonly impossible to detect. For these reasons, in all cases of severe knock to the head, we should act on the safe side, and give the parts as much rest as possible.

What may we find on post-mortem examination if death follows a severe contusion? (1) There may be a scalp-wound, or blood effusion in the scalp. A diffused blood-clot in the scalp tells us that there has been injury. (2) On sawing through the skull we may or may not find fracture. In some cases in which no fracture is apparent, one may be discovered on drying the bone. (3) If we find pus between the bone and the dura mater, and there is no sign of cancer or of syphilis, and no evidence of ear disease, it is a sequel of injury. (4) Pus between the dura mater and the pia mater, arachnitis, on one side of the brain, is also, in the absence of ear disease, cancer, or syphilis, evidence of injury. (5) The hemisphere underneath the pus is softened and yellow, through extension of suppurative inflammation. It is this cerebritis which kills the patient.

What is the actual suffering in such cases? Very commonly, after the injury the patient keeps about for a week or two, feeling uncomfortable in his head, but not thinking that there is much the matter. Then pain in the head and feverish symptoms lead him to seek medical relief. The pain increases, and is accompanied by confusion of intellect, tremor, and much restlessness. There is delirium, often epileptiform convulsions, and may be hemiplegia, or more general loss of power, and, finally, coma.

Inflammation consequent on Ear Disease.—Surface affection of the brain frequently arises from disease of the middle ear. Meningitis very rarely occurs in the early stage of suppurative otitis—that is, within a few weeks of the onset of the otitis—but it has been known to occur. Scarlatinal inflammation of the middle ear has, in the course of a few weeks, extended to the membranes of the brain and proved fatal. It is, however, generally in cases in which otitis has recurred over months or years that suppurative meningitis arises. With acute otitis there are very commonly symptoms indicative of much irritation of the nervous system, so that we are concerned about the brain. There is pain in the head, and often very severe giddiness, maybe a little delirium and tremor, with the temperature of the body raised. But these symptoms subside as the ear inflammation lessens, and we usually have to say that there are no signs that it has left any brain damage. Such attacks may recur over years, and the irritation of the brain may be very severe for a while, so that the patient lies in stupor for several days, and then recovers as the otitis subsides. When meningitis does set in, there is increasing severity in the symptoms day by day, the temperature remains high, there is increasing tremor, increasing loss of muscular power, delirium, and increasing tendency to coma. The suppuration commonly extends through the petrous bone to the dura mater in the middle fossa, and we may find pus on both sides of the membrane—that is, between the carious bone and the dura mater, and between this and the pia mater. It extends also through the pia mater, so reaching the surface and substance of the brain. In other cases the suppuration extends backwards, through the mastoid cells, and thence to the dura mater of the posterior fossa, and the suppurative meningitis, for such it usually is, makes its way towards the posterior lobes of the cerebrum or to the cerebellum. If the suppuration goes direct through the petrous bone, the middle portions of the brain are more commonly attacked. As the inflammation invades the cerebral substance, it softens it, and this inflammatory softening may extend as deeply as the corpus striatum and optic thalamus, and in this way bring about hemiplegia. The suppurative change may be thus widely diffused throughout the brain without there being any definite collections of pus visible to the naked eye, but in other cases there are such collections—one or more abscesses in the brain. Dr. Hughlings Jackson asked me one day, "Has it not been, in your experience, sometimes very difficult to diagnose cerebral abscess from more diffused non-circumscribed suppuration?" And I had no hesitation in saying that in some cases I had experienced that difficulty, and had been unable to make a differential diagnosis. We could not determine whether hemiplegia or other paralysis was due to suppurative diffused softening of the brain or to an abscess. In some cases suppurative inflammation extends from the mastoid cells to the dura mater, in and around the lateral sinus. Then pus is seen lying between the bone and the lateral sinus, the tissues of which are pigmented and softened, and the blood in the sinus is clotted, owing to the inflammation having extended to its lining membrane. This clotting may have extended along the sinus, and the clot have undergone a process of suppuration. Suppurative inflammation may thus travel down in the jugular vein, or along its outer cellular covering, until it reaches the chest. In this way may arise pleurisy, pneumonia, or pericarditis, with or without endocarditis, and kill the patient. In other cases a diffuse cellulitis may arise and extend down the neck; in others, a portion of the softened suppurating clot in the sinus or jugular vein may be detached from time to time and be washed into the lung, thus giving rise to patches of suppurative pneumonia, and in the end to numerous abscesses in the lungs. With each fresh outburst of pneumonia there is usually a very severe rigor. If, therefore, in a case of suppurative disease of the middle ear there are frequent and severe rigors, think it probable that the lateral sinus is plugged and there are recurring pyæmic abscesses in the lungs. If there are pain and tenderness down the neck on the same side as the ear disease, and also signs of pulmonary inflammation, this diagnosis is assured. Finally, let me mention that there are obscure cases in which suppuration in the middle ear leads to fatal abscesses in the brain, and on post-mortem examination we can find evidence neither of caries of bone nor of inflammation of the membranes of the brain. We suppose that the inflammation in these cases must have spread along a vein or a nerve-sheath.

Meningitis may also be excited by *growths* in the skull, cancerous or syphilitic. Idiopathic meningitis—a meningitis without any antecedent injury, without disease of bone, and without any morbid growth in or on the brain, is practically almost unknown. I have met with such a case, I think, only once. In this case the symptoms were like those of acute mania—pain in the head, acute delirium, increasing tremor, refusal of food, towards the end typhoid symptoms, and death from coma. We found most extreme arterial congestion of the meninges; they were coated with inflammatory lymph, which here and there was purulent.

Another form of suppurative meningitis, of which I have already spoken, is *epidemic cerebro-spinal meningitis. Suppurative cerebral meningitis* may also rise by *extension* of suppurative inflammation from the meninges of the spinal cord. Such suppuration may extend from the bladder or from near the womb. A lad was admitted to the hospital having lost the use of his legs, and he had signs of spinal meningitis, which extended until it became cerebro-spinal meningitis. He was very young, but I thought that the origin of the inflammation might have been gonorrhœa, for there was a history of purulent discharge from the urethra: but I found after death an abscess in the pelvis, and the suppuration had extended to the membranes of the spinal cord, and then travelled right up to the brain. After what I have said to you about idiopathic meningitis, you will understand that whenever you find meningitis you must look for some definite and well-recognised cause. The only two remaining forms of meningitis I have to mention are syphilitic and tubercular.

In *syphilitic meningitis* we are guided in our diagnosis not merely by the character of the symptoms but by their duration. Symptoms are usually repeated for months, or even for years, and the protracted nature of the disease itself makes us suspect that it is syphilitic. It comes on with pain in the head which usually disturbs the patient much at night; the pain is often severe and comes on in paroxysms, and associated with it are other symptoms of meningitis, increasing tremor, more or less delirium, giddiness, and very commonly repeated epileptiform seizures. If the change extends widely there is increasing failure of mind, and at last complete dementia. But it is wonderful how, in some of these extremely demented cases, the mind after a while becomes clear and active again. As the syphilitic meningitis extends it thickens the meninges; the lymph becomes organised and passes into a fibroid substance, and this becomes firmly adherent to the surface of the brain; the dura mater, the pia mater and the brain surface are bound together. Amidst the fibroid material we find one or more wash-leathery looking syphilitic nodes; in the brain, as in the liver and other organs, there are two types of syphilitic change, cases in which there is much gummous material and little fibroid change, and those with much fibroid thickening and very little gummous formation. The latter are the worst cases. The syphilitic fibroid thickening frequently extends into the substance of the brain, leading to induration of a portion of the hemisphere, and it may be to permanent hemiplegia. Syphilitic gummata are very rarely met with in the basal ganglia, but they may occur there, and also in the cerebellum and pons.

In some cases intra-cranial syphilitic disease may be most marked in, I will not say limited to, the sheaths of the cranial nerves; the third, fifth, and sixth are those most commonly affected, but it may be the seventh, causing deafness or facial paralysis, or one of the others. In other cases the arterial structures suffer from syphilis. For instance, a small gumma may form in the adventitious coat of the middle cerebral artery, and later extend into the inner coat, until the blood coagulates in the vessel, and so leads to softening of the corpus striatum and consequent hemiplegia. I have known thrombosis arising in this manner extend across to the opposite middle cerebral artery, so that the patient died with softening in both hemispheres. These are cases in which hemiplegia comes on rapidly, commonly in youngish subjects, and there is no heart or other disease to suggest embolism.

Some years ago the mental symptoms associated with syphilitic brain disease led asylum physicians to speak of them as "syphilitic insanity;" they considered that the symptoms were not only distinctive but of a kind limited to syphilis; but of late years they have come to see that they cannot distinguish such symptoms from those of other cases of insanity. In some cases there may be distinct features of syphilitic disease, and yet other causes may have been in operation—for instance, a patient has signs of recurring meningitis, and there is a history of syphilis, but there is also a history of a severe blow on the head.

In some of the cases the symptoms are beyond question due to syphilitic disease, but in other cases the diagnosis is exceedingly obscure.

A little while ago this experience happened to me. A poor fellow had had syphilis, but he had been much worried in his affairs, and he had had much anxiety about his family. He had a maddening idea of syphilis. I was asked to decide whether the insanity was the result of money worries or of syphilis, and of course I could not. What I labour to impress on you is, that we have to bear in mind that these symptoms which seem to denote syphilis may have other origins. I saw another man who was starving to death under fear of syphilis. In that case there were symptoms pointing to spinal cord and brain disease, and I could not say that they were not due to syphilis, but there were other causal conditions.

It has long been recognised that, especially at the middle and later periods of life, after many years of toil, worry, extreme exertion, and mental anxiety—until at last the anxiety has become almost unbearable, and common events are madly felt—that in such cases of brain disease there are slow degenerative changes in the meninges of the brain, rendering them more opaque, thicker and milky-looking; and this thickening extends along the vessels which supply the convolutions with blood. This thickening is spoken of as the result of chronic meningitis. and in its more marked forms we call it pachymeningitis. It is distinguished by recurring acute and sub-acute attacks of brain irritation, pain in the head, vertigo, exceedingly harassing dreams, and other more or less delirious disturbances in the mind. It is difficult in some of these cases to distinguish if syphilis be a deteriorating agent or no—but when a poor fellow is going mad, to saddle him with a fear of poison is, to say the least, torturing—he becomes haunted with the fear of being poisoned. This form of pia-mater thickening is met with in various forms of insanity.

Tubercular meningitis is one of the commonest forms of meningitis. When looking for the cause of meningitis, always first notice whether the morbid deposit is most accumulated at the vertex or at the base; if at the former, the meningitis is not tubercular. Tubercular meningitis is always most marked between the optic commissures and the pons Varolii. We notice that the pia mater is no longer translucent and clear, but is opaque and exceedingly hazy from accumulation of yellowish-grey lymph. The lymph may be very grey, with a good deal of serous effusion, but the cloudiness of the pia mater is a constant appearance. A similar change extends along the fissure of Sylvius, also along the posterior cerebral arteries into the lateral ventricles. We trace the effused lymph along the course of the arteries, and the lining membrane of the ventricles may be coated with lymph. The change may travel backwards over the pons, but does not usually extend far in this direction. When we see such appearances we look for tubercle. In some cases tubercle is evident to the naked eye, but in others can only be found on microscopic examination. Even if we find no tubercle on naked-eye examination we are confident with such appearances and distribution of inflammatory changes as I have described, that the meningitis is tubercular. Most commonly, however, with the naked eye we see a number of minute grey bodies scattered along the track of the vessels. If we take a little piece of pia mater and float it out in water the tubercles can be seen more distinctly, and still more so on holding the membrane up to the light. With the microscope on following up a small arteriole we may find that in places its wall is bulged out, and that there is an accumulation of corpuscles in the adventitious coat. This is a tubercle. We find also that the ventricles are very full of serum—acute hydrocephalus this disease used to be called. We further notice much softening of the wall of the ventricles, but without the appearance of inflammation—this is post-mortem softening. Owing to the accumulation of fluid in the lateral ventricles, the surface of the brain is jammed against the skull, and hence the convolutions are flattened and the circulation in them brought to a stop—in this way death is brought about by coma.

What is the suffering in tubercular meningitis? You will easily bear in mind the symptoms if you remember what I have said about other surface diseases of the brain, and how in this disease death is brought about by coma. Antecedent to the onset of the meningitis there is loss of flesh, such as is characteristic of the tubercular diathesis. The antecedents guide us as to the kind of meningitis. There is progressive weakness and anæmia. After a time, it may be months, of preliminary failure of health, signs of peripheral irritation of the brain set in. There may be feverishness, but in some cases that is little marked; the characteristic symptoms are pain in the head, restlessness, peevishness, frequently repeated retching and vomiting. Soon the symptoms markedly point to meningitis—there is increased pain in the head, delirium, but not much, differing in this respect from maniacal forms of meningitis; there is increasing tremor, the pulse becomes slow, the breathing labouring and irregular, the cheeks are flushed, there is the piercing cry of cephalic irritation, and convulsions may come on at any period of the disease. I have known epileptiform convulsions at the very outset. The vomiting is most marked in the earlier stages of the disease. To the signs of peripheral irritation gradually succeed those of cerebral compression. After about a week the pulse becomes more rapid and irregular, the breathing becomes more irregular, being sometimes very quick, sometimes slow; the delirium is replaced by increasing stupor, the restlessness by powerlessness; the body is starved, the abdomen is sunken, the cheeks are sunken, the bowels cannot act, the limbs cannot move except by tremor or violent convulsion, and after another week or ten days the coma passes on to death.

I have yet to speak of two or three more surface lesions, and we will first consider *meningeal hæmorrhage.* By this we mean an effusion of blood between the dura mater and the pia mater —this is at least the usual form of meningeal hæmorrhage, the blood coming from one of the meningeal arteries. The cause of the bleeding may be aneurysmal dilatation leading to rupture, or other degeneration such as atheroma or fibroid change, making the vessel brittle and easily ruptured. In other cases the effusion of blood may be due to morbid change in a superficial branch of middle cerebral artery, or in rare cases of the posterior or anterior cerebral artery, and in such cases there will be effusion of blood both within and without the pia mater. In such cases hæmorrhage may be profuse and rapidly fatal, and at the post-mortem examination we may find a layer of blood-clot a quarter of an inch thick spread over the whole surface of one hemisphere.

If you are asked, "Does intra-cranial hæmorrhage ever kill suddenly—in a few minutes?" answer, "Yes, and in such cases we may find one of two conditions—either meningeal hæmorrhage, or hæmorrhage into the pons Varolii—other forms of cerebral hæmorrhage kill more slowly." More common than sudden death in cases of meningeal hæmorrhage is the onset of epileptiform seizures; there may be repeated fits, and the patient may then rally or die.

There is a third group of cases of meningeal hæmorrhage in which the symptoms come on more slowly still. The patient is confused and staggers, and we wonder if he is drunk or shamming. In speaking of convulsions I told you how a patient with meningeal hæmorrhage was treated in the receiving-room with the battery. Such cases are not uncommon, the patients being either believed to be drunk or to be shamming. They are apt to be violent and abusive, and I remember one patient from whom the only remark we could get was "Damn you." These cases often find their way into the police cells, and there rapidly become comatose and die. In other cases, but these are much less common, the patient walks about in a confused state for a week or two, and then there is an acute outburst, and he dies.

If the patient survives many weeks or months, instead of finding, as in recent cases, a blackish-red coagulum over the hemisphere, we find the rusty red remains of the blood-clot. If death occurs at a still later date we find that a cyst-wall is forming around the old blood-clot, and this may be loose, or it may be adherent to the membranes; the blood-pigment has been much absorbed. Finally, a cyst filled with more or less serous fluid remains on the surface of the hemisphere. A cyst of this kind, resulting from either traumatic or spontaneous meningeal hæmorrhage, may lead to recurring fits, pain, and giddiness for years.

An *aneurysm* as large as a pea or a small nut may form on the surface of the brain. It may be connected with the middle meningeal artery at the base of the brain, and press on the third, fourth, fifth, or sixth nerve. A tumour may be believed to be present, and, in addition to the usual symptoms of tumour, the patient may complain of ticking, whirring, and pulsation in the head, but no certain diagnosis can be made.

TUMOURS OF THE BRAIN.—*Medullary carcinoma* at the base of the brain causes somewhat similar symptoms, slowly invading the nerves, and leading to paralysis. There is not usually much pain. We can diagnose a growth at the base of the brain, but cannot specify its nature unless the glands become involved.

Glioma is another peripheral growth. It always occurs near the membranes of the brain. We commonly find it invading one of the convolutions, and pressing the pia mater outwards. It may have begun in the crus cerebri or in the optic thalamus or corpus striatum, but in these regions also we find the growth close to the pia mater. Gliomata are tumours of nerve tissue—brain tumours. They occur also in the retina, and perhaps also in the spinal cord. The appearances vary according as the disease has killed the patient in few or in many months.

If the growth is very recent we may easily overlook it, as it so much resembles in colour the convolution itself. It is very soft and jelly-like, and we are immediately struck by its bluish grey translucent appearance. It has no abrupt line of demarcation, but blends with the tissue. These tumours are very vascular, so that on section we often see scattered red points. In some cases they are extremely vascular, and bleed so much that at first sight we mistake them for blood-clots. There may be two or three growths. Under the microscope we find that the tumour is made up of nuclear bodies with long tails—a kind of thin spindle-celled growth with dilated vessels, an active growth. If the tumour is older it is much firmer, and more or less fibrous, and it may have become more or less yellow from fatty degeneration, but surrounding the firmer fibrous material there is some of the bluish-grey jelly-like substance.

The growth is peripheral, and hence the symptoms are those of surface irritation. There is increasing pain in the head, and epileptiform seizures are common. In other cases there is increasing mindlessness. The patient becomes duller and duller, and more or less sleepy. "He goes to sleep even while he's eating," say the friends. Slowly coma comes on. When the growth is near the base of the brain the nerves are affected. In nearly all, if not in all, cases there is optic neuritis.

The disease occurs mostly in young persons—it is certainly rare beyond middle age. If the growth is very slow the symptoms may be exceedingly obscure. A patient was taken into the hospital suffering from phthisis. While walking in the ward he suddenly fell down. It was then noticed that there was ptosis on one side. He rapidly became comatose, and died. At the post-mortem examination we found a glioma in one crus cerebri. His friends were sent for, and asked if he had suffered much in his head, but they thought not. The medical officer, the nurse, and the sister, had seen no brain symptoms. All we could gather as evidence of brain trouble was that one of the patients said that the man a day or two before his death had been unable to open one eye.

We sometimes meet with *strumous formations* in the brain. Sub-acute inflammation occurs in the pia mater, most commonly in the cerebellum, but it may be in the posterior parts of the cerebrum. This inflammation leads to œdema and exudation, and the exuded matter degenerates into a caseous substance. Strumous tumours of the cerebellum may remain latent for months or years.

We often find *cancerous secondary growths* in the brain, and one or several similar growths in the liver. In the brain, just as in the liver, they soften down in the centre, so that in some cases we find cavities filled with fluid, but there is always a margin of medullary cancer. These cases also are often very obscure. One of the sisters in the hospital died of this many years ago; the case for a long time was most obscure, but she suddenly became blind.

DISEASES OF DEEPER PARTS OF BRAIN.—In diseases affecting chiefly and at the outset the substance of the brain, the symptoms are not, as in surface diseases, chiefly those of irritation, but are those arising from loss of volitional power. When pain and delirium, and other signs of surface irritation arise, it is owing to the extension of morbid change to the surface, or more commonly to stretching of the pia mater. As the pressure increases, and the convolutions become flattened against the skull, the pain and delirium may increase, but there are increasing signs of paralysis. There are commonly epileptiform or apoplectiform seizures, leading to coma. But at the outset let me repeat that the symptoms are usually those of loss of volitional power and lessening conscious expression. This is illustrated in cases of abscess in the brain and in cerebral hæmorrhage.

Abscess in the brain may be due to pyæmia. In some cases the abscesses are numerous, so that the brain is much destroyed, and there are also abscesses in the lungs. In other cases there may be a single pyæmic abscess in the brain secondary to old abscess of the liver. In other cases we find one or more abscesses in the brain consequent on disease of the middle ear, whilst in others abscess is a sequel of contusion.

When examining an abscess in the brain note the condition of the abscess wall. If it is exceedingly thin and soft, the abscess is recent, dating at most from three or four weeks back; in such cases the pus is yellow, what is called "laudable pus." If the abscess is older, the wall is thicker and very tough, and the pus is greenish-yellow and ropy, having degenerated into a gluey material.

In some cases abscess kills the patient rapidly. As the seat is usually in the white matter, pain only comes on when the pia mater is rapidly stretched by the accumulation of inflammatory material. If this occurs, there is pain persistent night and day, giddiness, epileptiform seizures, and, towards the last, hemiplegia or general paralysis, and coma. The patient may be able to walk about till within a few days of his death, with much pain in the head, but no fits and no paralysis.

If an abscess forms slowly, so that no rapid stretching of the tissues occurs, the symptoms are extremely insidious. For instance, a man falls from a haystack on to his head; he is stunned for a while, but it passes off, and he seems to be well. After he has been at work again for months, suddenly cerebral symptoms come on, and the patient dies of abscess of the brain.

In *cerebral hæmorrhage*, if the hæmorrhage be small, and occur away from the spinal tract and the basal ganglia, and away from the pons—say, in the centre of the hemisphere—the symptoms may be few and obscure. In some cases there is a sudden interruption of some volitional act, varying according to the position of the hæmorrhage. It may be a sudden shock or stoppage in speaking or writing, or a slight temporary attack of paralysis in one arm or hand, or a sudden fall, in which the patient does not lose consciousness and the reason of which he cannot understand, or there may be a slight convulsive seizure. This disturbance may pass away in a few minutes or hours, and the patient seem to be well again, but such small hæmorrhages as are evidenced by these symptoms are commonly the precursors of larger ones. We have seen their remains so often in the brain after death from cerebral hæmorrhage, that we are inclined to think it probable that there are always lesser warnings antecedent to major seizures, though the lesser are often overlooked.

Cerebral hæmorrhage is most common in the corpus striatum and optic thalamus; next to that in the pons; it rarely affects the cerebellum. If it be large it breaks up the striatum or the thalamus, often much of both; and the blood also commonly makes its way into the lateral ventricle, and breaks thence into the ventricle of the opposite side, so filling both. It also commonly bores down the crus cerebri towards the pons. In other cases the hæmorrhage extends more towards the surface of the hemisphere, having arisen from the outer part of the striatum or thalamus. In these cases epileptiform convulsions commonly usher in the seizure.

Cerebral hæmorrhage may occur during sleep, and nothing be known about it until the patient wakes. In other cases there is at the outset a kind of shock, a sense of sudden confusion, which has been called cerebral surprise. That is recovered from, and then, after some minutes or hours, loss of volitional power becomes marked. After the onset of hemiplegia there is usually for twenty-four hours a period of calm, but soon, as inflammation occurs to some extent in the brain-substance around the clot, the temperature rises. This is the so-called "cerebral fever." Coma may come on from the pressure of the accumulating blood in the earlier period of the seizure, or later from the softening or swelling effects of the inflammation. If the hæmorrhage be very large and very rapid, the symptoms may in a few minutes from the outset be those of coma. The increased stupor that comes on during the course of the cerebral fever is very commonly recovered from. In cerebral hæmorrhage there are two great risks—the risk of compression of the brain, and the risk that the extravasation may penetrate as far as the medulla and stop respiration. We therefore measure the severity of an attack by the degree of persistent coma, and by the degree to which the breathing is impaired. What we always aim to do in cases of cerebral hæmorrhage is to maintain the circulation and breathing.

Here let us ask, What do we mean by *coma?* We mean that the deeper conscious activities of the mind cannot communicate with the outer world. You may prick the skin, or injure it more severely—the patient does not manifest any feeling. You may irritate the conjunctiva—there is no reflex manifestation of sensibility. You may make a loud noise—there is no sign of hearing. Should the compression be removed, the breathing and circulation become more natural and consciousness returns—the coma passes away. What is death by coma? As the brain becomes compressed and its function excessively interfered with, the breathing becomes more and more labouring and the lungs more and more congested, and death results from failure of respiration and breathing. I have found on examining the lungs that the air-cells are choked up with yellow corpuscles—there are, of course, some colourless, but mostly yellow. The tension of the pulmonary vessels had increased to such an extent that they were unable to hold their blood.

If the hæmorrhage has been small in amount and death has occurred in a few days from its onset, we find a blackish-red blood-clot in a cavity of broken-up brain substance. There is no cyst wall. Around the cavity we find a number of little red points of blood-clot and ruptured and plugged blood-vessels. Where the hæmorrhage has been larger we find the lateral ventricles filled as I have already described, and the blood may have passed into the third ventricle, and thence under the pia mater into the fourth ventricle. The brain is stretched and the convolutions flattened. If a patient who has had a comparatively small hæmorrhage dies weeks or months after its formation, we find, if the time is measured by weeks, a mass of rusty red colour. After a few months a cyst only remains, or a quantity of hæmatin or hæmatoidin of an ochrey-yellow colour, and fibroid tissue in which the nerve tubes are buried. There is, in fact, a scar in the brain.

Softening of the brain has been divided into "red," "white," and "yellow." Red softening is usually due to embolism, white more commonly to thrombosis. Red softening is more rapid, white softening slower. Red softening usually occurs from embolism of one of the branches of the middle cerebral artery near the surface of the brain. We find a portion of the brain very soft, and of the colour of blanc-mange mixed with red-currant jelly. More than one artery may be plugged. The degree of redness is a measure of the acuteness of the attack. If, after the plugging of the artery, some time elapses before death takes place, much of the effused blood-pigment is removed, and we then find an area of what is known as yellow softening. We meet with cerebral embolism mostly in connection with old rheumatic heart disease, but it may arise from other causes. In thrombosis there is a slow formation of blood clot on the lining membrane of a diseased vessel, atheromatous or syphilitic, and the brain crumbles down into a white, pulpy, worm-eaten mass.

There is another class of brain diseases in which there is fearful suffering, *acute mania* and *acute melancholia*. They are really more fearful than the gross organic changes of which I have been speaking, and yet we find so little on post-mortem examination. We may fail to detect any changes in acute mania. In acute melancholia there may be serous effusion under the pia mater and nothing more. In many cases of suicide evidently due to insanity, or in cases of sudden apoplectic seizure, or in cases of general paralysis of the insane, we may find milky fibroid changes in the pia mater, and the convolutions of the anterior lobe atrophied to some extent, and serous effusion under the pia mater. Or there may be a little thickening of the pia mater and a few old adhesions of the membranes—nothing more. But we very commonly find similar morbid changes with no such clinical history of insanity, and we therefore infer that the morbid change connected with acute mania and acute melancholia lies much deeper and affects much finer workings.

General Considerations and Treatment of Nervous Disease.—The elements in the living body, the water, the serum, of the blood and the tissues, cannot be separated from heat, and life, and mind. We must remember that when our aim is to relieve the suffering of the individual we must not leave his mind out of count. Then we have no longer to deal with morbid physiology but with morbid psychology. In studying pathology we have learned that there may be much organic change, and yet the individual may suffer very little. We see this in diseases of the liver, kidney, lung and brain. We have also learned that there may be very great suffering with little appreciable morbid change.

All disease, as such, must have a relation to psychology. I go so far as to say now, " Disease and madness are inseparable." Immediately we are in disease we take a wrong view of the world. I know I always do myself—when I am ill the world becomes gloomy and sad.

We have to aim at keeping our natures one and undivided, then we are wholesome and healthy. We must carefully draw a distinction between healthy psychology and morbid psychology. Healthy psychology always goes for rest, and health, and peace—in confidence and sweetness it loves to enjoy. That is love of life. Morbid psychology always goes for separatedness. When insanity is coming on, it is always some special organ that is complained of—it may be the bowel, the stomach, the womb, or the bladder. It is always something special, no longer the one and undivided nature.

Now let us bestow a little more time on a most important question. How is it that the structures with which volition most works become most paralysed? Why, to repeat the question in another form, do the organs of volitional expression suffer so much from disease? In various ways the natural tensions of these organs are interfered with and destroyed. Let us notice the trembling and increasing weakness of on-coming paralysis of the arms and legs, whether due to morbid changes in the spinal cord, or to affection of higher tracts, the corpus striatum or optic thalamus; or the atrophy of cerebral convolutions, leading to volitional paralysis; or the progressive muscular atrophy of the hands and feet; or rheumatic gout changes, with bony outgrowths and atrophy of cartilage in the joints of the hands and feet; or the morbid changes in other organs of expression—the ocular muscles, the vocal organs, alike of larynx, lips, and tongue; all these structures of volitional expression are prone to suffer from disease. The automatic, more vital, actions, escape disease much longer. Why is this?

The familiar answer might be that the will is not disciplined enough—there is too much liberty, and that leads to restraint—immoderate use leads to loss of power—the extreme of self-will drives to utter madness—disease being the expression of madness in the several members of expression.

But how comes the immoderate use, the too much liberty—libertinism? To answer this question, let us consider that the natural movements of the organs of expression—arms, legs, and other organs—are all rhythmical, for they are all dependent on the rhythmical influence of the circulating blood, they are governed by that circulation. When that rhythmical circulation ceases they are completely paralysed. It is demonstrable that the organs of expression act rhythmically. It is witnessed in running, in singing, in successful workmanship, in the movements of children and of animals; we may see it by daily observation; in fact, a limb working is a compound of many rhythms, and rhythmical working is easy working.

But in the diseased operations of the organs of expression there is not ease, there is increasing difficulty, until at last there is paralysis—loss of rhythmical strength and activity. How comes that failure? The will, the volitional power itself, becomes diseased, and that is the result of restlessness. The mind is not allowed to go year by year restfully, in the great natural ways, operating with the power that is willing the growing and development of living things. Will is persuaded to take liberties, persuaded to restricted thought. Self-consciousness, so-termed, not reason, leads the will to prefer and to take, day by day, the difficult rather than the easy. We do that best which we do easiest; if we try the difficult, we may go on straining at it until it becomes diseased and paralysed. The strain interrupts the rhythm of ease. All this is demonstrable. It is not the use of free will which causes disease; that is free which is freeing, which enables us to go freely onwards, not restraining and paralysing; it is enslaved will which causes disease.

Here, however, we must not stay too long, but we must go on to inquire how the nutritive changes of the diseased structures are to be guided to health again. To learn that, let us ask a question which is the complement of that we asked before. How is it that the automatic movements and structures longer escape disease? Briefly, I will answer that it is because they are under the influence and government of the elements—of the great servants of the Supreme Will. They are under the influence of the air entering by the lungs and operating on the skin, of the food and water entering by the stomach—in short, of all the elementary influences which go to make up the circulating blood, and they are governed by energising, unconscious impressions which enter the nerves of the skin, maintaining human breathing and circulation, as animal and vegetable functions are maintained. The skin structures may be irritated, worried, and weakened, until the automatic functions fail. This has been already explained earlier in the lecture. We learn that by automatic we mean more natural, more in accord with Nature at large, less under our pretensions and professions.

There is another form of expression besides the volitional—the getting out of the body of the used-up effete material, the excreta. With diseased conditions that form of expression usually fails; there is failure of excretory function, until the excretory organs are charged with morbid material and rendered useless. The excretory (expiratory) function of the lungs fails, and the air-cells are filled with tubercular material, or the air is comparatively stagnant, as in vesicular emphysema, and the blood poisoned by carbonic acid. The kidney excretory function fails, and the convoluted tubes are choked up or otherwise destroyed. The liver excretory function fails, and the liver cells become compressed and destroyed. The excretory function of the bowel fails, and the rectum may become strictured and choked up with cancer or other morbid accumulation. The skin excretory function is interfered with, and eruptions of various sorts clog the skin, until it loses the privilege the crab and snake possess of casting off their outer covering in time and season for beautiful and healthy progress.

To obtain a view as to how that failure of excretory power is brought about, let us recall what the physiologist teaches us, that the inspired oxygen comes in contact with the plasma and combines with it, so releasing the used constituents, which pass down into excretory products. As far as it goes we can accept his view, but we have further to ask, What is the nature of the proceeding that takes away the used-up material? It must be energy of some kind. It would not do merely to oxidise—there must be a power to get the result of the oxidising process out of the body. We have to ask, What is the power in natural proceedings which takes away the effete material? I have said that its setting out is often hindered until the body is destroyed. The accumulation of effete material is more deadly than living death, for in living death the plasma takes form in time and season, and keeps it only so long as is consistent with the easy progress, the working further into life; but these accumulations arrest the progress of the body, leading it, evidently in many cases prematurely, to the grave.

In natural proceedings there are constructive and destructive powers, and human nature being, as biology teaches, the consummation of the evolution of all other nature on the earth, we might fairly expect to find both powers acting evenly in us in health.

You hear me remark sometimes, that, to me, it was a great discovery when I found that I had a profound respect for rot—for spring comes by rot. The severe but helpful operations of winter rot the weaker living material down, to promote the survival of the fittest, and as the sun-energy comes into spring, swarms of organisms begin to stir about and take away the *débris*, and with the increasing heat, broods of active creatures further put the living plasma of plants and animals to the severest tests—all to engender by increasing power. It is thus traceable, that nutrition is a struggle of opposites, and that the world's progress is secured by the loving law of necessity, for in this law is obedience to the God of power. Such is ease. It is this circulating power working to promote clear definite ends, which executes and performs the functions which make structures. The proof of this is the fact that, when circulation ceases, there is no function, and the forms disintegrate to the formless again. Disease is due to the natural circulation being interfered with, and so the accumulations collect which are more deadly than the active destructive processes which may be called living death.

We have remarked that the circulation is maintained by the great respiratory power, working through our natures to things of the earth, making all one.

We may pertinently inquire if there be not the same necessity in human nature as in animal and vegetable nature, that, by the aid of contact and contention, the energies of the vegetable and animal world are transmitted to us, and those of our nature to them—they pulling us down while we pull them up—this showing the justice of even-handed proceeding, all handsome in doing. Again, the ease of the world's progress—such it seems to my thought—may be the great use even of "germs" and "bacilli" in us—an instance of how it is we cannot escape in healthy proceeding contact with rot and other contention.

And it would seem that if any of us ignore our animal and vegetative functions, we are ignoring the constructive powers of our nature, until the destructive powers have it too much their own way, and at last the lowest take our remains. This reveals how conventional restricted views, ungenerous, unkind, limited notions, bring about madness and disease. Too much indoor proceeding, too much flattering unction, with its wearying and killing mistrust, take away much energy, and give little or none in return. But whatever is thought about rot and other destructive powers, it cannot be denied "that all things are beautiful and decent in the true return of their seasons."

Here, gentlemen, I must begin to cease. I am tempted to say as Raleigh said, "Fain would I climb, but that I fear to fall." I would climb more into the consideration of disease, but the time allotted me forbids. The beautiful, but mysterious mistress Nature, ever engendering progress, warns me that I cannot now do it boldly, so I had better not do it at all. The more I court Nature, the more I see that it is meant that we should know, the more I see that it is given to every one of us to know; but to reach that, we must believe in Nature's ways—we must believe in what is in us, must believe, that whatever theories may be put upon us, with whatever explanations we may be saddled, and whatever discouragements are thrust upon us, there is a marvellous order and power for knowing working in us all. I often feel that I should like to take the students and with them sit upon the earth naked, to know—to feel with the great mind knowing and making all things—to go the way to get our senses free into Nature's widespread operations, and to enable us to be a unity with the One. I am the more urged to do so, by noticing that policy has not a happy life. It is the oneness that we have always to keep in view; it is everywhere before us—the sands of the sea are millions of ones, the plants in their growing, the birds in their singing, the children wending their way on the other side of the shore. The accountant casting up his rows of figures inculcates that he is lost if he loses sight of the unit—that all his figures are units united. With us, if tubercle, or suppuration, or fever, mars structures until, by their differences, they become abnormal and separate, we have to lead them back to evenness again—ease being the smoothness of energy.

Ideas hinder that, for they are too hard and fast, too much form without energy—they are abstract. When the human mind was endeavouring to come out of the darkness of the Middle Ages, away from abstractions and disease, and was striving to become more capable—when human thought struggled to get free from clogs and enter the Reformation—there was an intense craving and want for some other guidance than that of mere ideas and tradition. Men longed to be amongst those who could sail o'er the sea and could live with the elements, and wander more usefully and powerfully over the earth.

Following that upheaval came Locke's "Essay on the Human Understanding," in which he taught, on old lines, that human creatures learn to know by the use of their senses. In so doing he further opened a way to get a basis of fact for the outer world's co-operation with the mind; he made exact science more attainable, and showed how truth—how the things that are—could be demonstrated.

It was perceived by Descartes that we must seek further than this, or be lost. Asking himself: "How is it I know by my senses?" he answered, "Because human nature is endowed with a thinking power;" and the proof to his mind of God was the fact that he could think so. The proof was in the possibility, and possession was in thinking. The Almighty is working in and making everything—there is mind in everything. Stability lies in the continued making of all things—certainty and indestructibility lie in continuous ordered changing. As Spinoza said, there is but one substance, that is God. This substance is the oneness in human and all other things derived from the Eternal One—the Almighty energising, giving ease to all Nature, and eternal rest by the freedom in progress.

So by the nature of things we may recognise ease as health, and its sweetness in feeling as happiness, and as pathologists we have endeavoured to trace how ease is lost and human nature diseased. No course short of that could enable us as medical practitioners to afford the required relief—to help the patient to recover health again.

The world at large demonstrates that as things are happening they are happening for the best ends; making the most; feeding the largest number; progressing ever; and in that happy state of things is a renewing, reconstituting, healing, healthy power—the vis medicatrix naturæ. But it must be seen, Providence is not fatalism; it is intelligence—seeing detail by detail. Singlemindedness is clearmindedness. Therefore, to secure clearmindedness we must keep before us the unity of Nature, and for this it is well to remember what those taught who have taught us to devote ourselves to the one God. "Bless God for the evil as well as the good. There is but One" (Talmud).

To do that we must, to the utmost, endeavour to do our best, and take all as it comes; the renewing, healing, circulating; to avoid useless regrets and vain anticipations; to be present in the timed operations of things. We must endeavour to promote the interests of our individual patients, recognising that every individual must rest according to his own temperament, and knowing that the welfare of society is dependent on the well-doing of its individual members. But daily its members are tempted off by passing appearances; illuded, bewitched away, they cannot hold their own natural course enough; so the required progress and ease are hindered and lost—hampered by narrow views, little thoughts and ideas, and by too restricted impressions—until poor self has a hard time of it. Miserable in its loneliness is poor self; nowhere sufficiently understood, because led off to be too superficial; sensuous, because not feeling enough its way to perceive, to reason, and so to know how to go for happiness—its course may be expedient, convenient and conventional, but for what interests is it adapted?

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